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ON ABDOMINAL INJURIES AND THEIR RECIPIENTS*

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THE most prolific cause of abdominal injury throughout the centuries, even back to geological time, has been the warring instinct of mankind: historical museums indeed display portions of the human lumbar spine with impaled flint arrow-heads; some of these relics embedded in the anterior portion of the vertebral bodies (*Fig.* 152) limn with unerring surety the transabdominal course of a missile shot long ages ago by some enemy hand of the Neolithic period, a dual monument to victor and to slain.

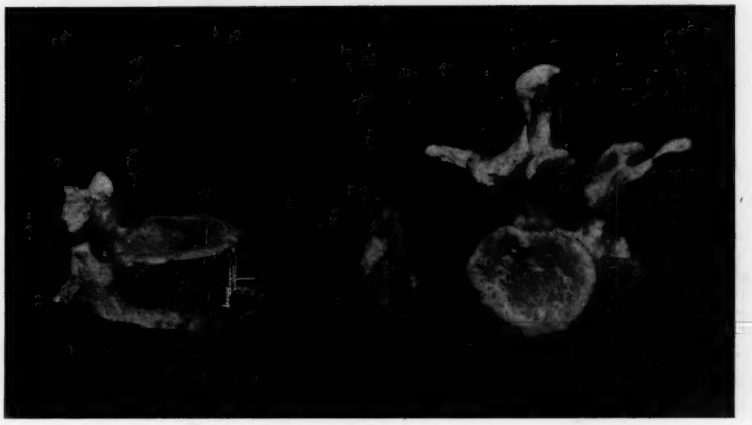

FIG. 152.—Lumbar vertebræ with infixed flint arrowheads (casts). Neolithic. La Grotte de Villemard (Marne). (Originals in the National Museum, Saint-Germain-en-Laye.) Also an example of a flint arrowhead. (*By kind permission of the Wellcome Historical Medical Museum.*)

When combatant armies fight at a distance from each other with all the machinery of the modern battle, injuries of the abdomen are nowadays no more frequent than those of other regions, and constitute some 2 to 3 per cent of all the injuries of warfare; where proximity between foemen becomes closer, the belly constitutes a target, and the tale of abdominal wounds must consequently

* A fragment of an Address delivered to the Academy of Medicine, Toronto, on April 5, 1938.

increase. Ferracute, the giant Saracen enemy of Charlemagne and his Franks, claimed his abdomen as immune to injury, except at the navel—a veritable ' bull's eye ' ! The boast proved his undoing, for Orlando, engaging him in single combat, stabbed him in the umbilicus and mortally wounded the Goliath.[1]

" Captains and kings " have fallen in battle with abdominal wounds : sometimes " their name liveth for evermore " : thus Olaf, a Norwegian sovereign, has become patron saint of his kingdom. The memory of others has remained evergreen in the proud and grateful hearts of their countrymen : Wolfe (*Fig.* 153) and Montcalm each perished from injuries to the belly, and the story of Quebec remains an epic for all time.[2] A few, like William the Conqueror, have perished of an intestinal injury received under less heroic circumstances ; the bowel was damaged, and that

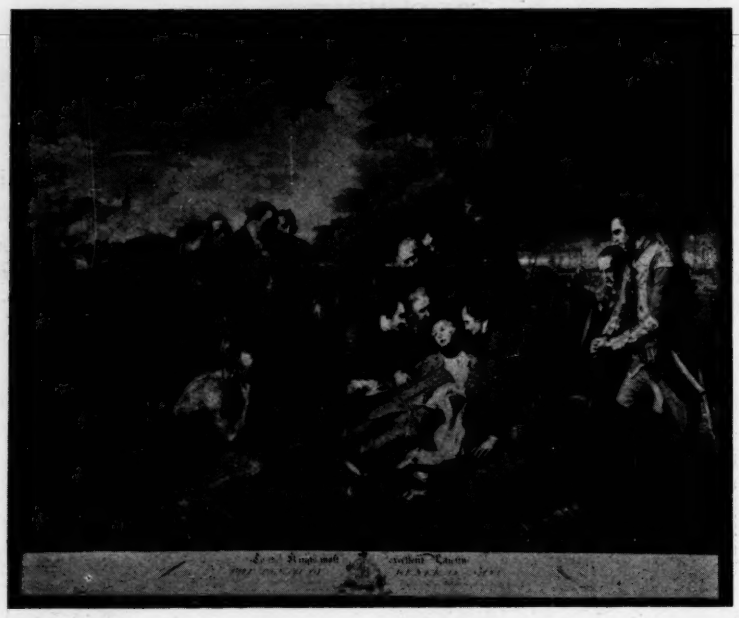

FIG. 153.—The death of General Wolfe. The application of pressure over Wolfe's left hypochondrium is depicted. (From an engraving by Wm. Woollett, Engraver to His Majesty King George III, from the original picture in the collection of Lord Grosvenor.)

monarch's death subsequently ensued, when his hernia struck the tall iron pummel of the saddle of the horse that he was riding during the destruction of Nantes.

Even before the days of Horace, patriotism had inspired the fine sentiment—" *dulce et decorum est pro patria mori* " ;[3] every schoolboy in the kindergarten knows how Decimus, Mus, and many others " devoted themselves to death," when victory for the Roman arms still hung in the balance. Priests and worshippers professing certain creeds or faiths who die in battle look forward with confidence to a piece of carpet and a select harem in the hereafter ; this appeal to the passions adds zest to their warlike qualities, and in days past the body and belly of the Asiatic and African furnished an easy target for the European bayonet.

Some whose names are inscribed on the Roll of Honour, when mortally wounded in the abdomen, have learned before death that their gallantry has been rewarded by the highest badge of bravery ; of others the " Cross " has been a posthumous consolation for sorrowing parents. Young Roberts, emulating his

distinguished and beloved father in his V.C., was mortally struck in the belly by a Boer bullet at Colenso, and Chavasse, earning the most prized decoration of the British Army for a second time, died behind Ypres of an abdomino-thoracic injury.

The cherished emblems of gallantry and self-sacrifice may not always be those bestowed by a sovereign; the Sam Browne belt of a regimental medical officer, with significant perforation due to a piece of high explosive, was long prized by a loving elder brother, himself a surgeon, thereby reminding him of the fortitude and all the other virtues extinguished for ever by an abdominal wound, which he and other eager and anxious surgical friends could do nothing to cure.

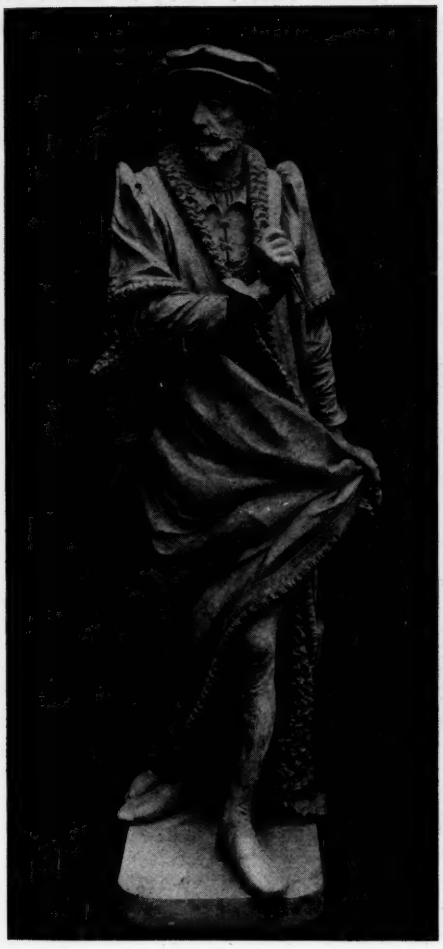

FIG. 154.—Sir William Walworth, Lord Mayor of London, who slew Wat Tyler in 1381. (*By kind permission of the Prime Warden and the Court of the Fishmongers Company.*)

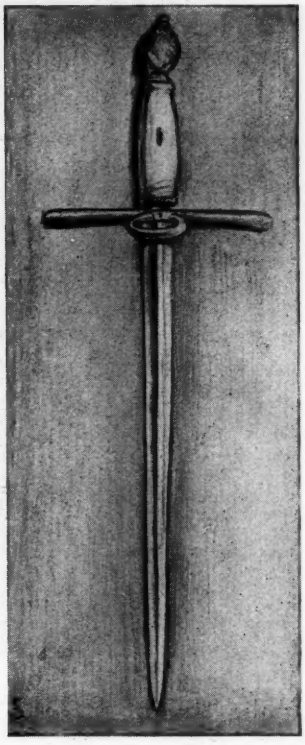

FIG. 155.—With this dagger Sir William Walworth, Lord Mayor of London, slew Wat Tyler in 1381. (*By kind permission of the Prime Warden and the Court of the Fishmongers Company.*)

Organizations, military and civil, make provision for substitution or succession; Penn-Symons, commanding the troops at Talana Hill in the South African War, was fatally wounded in the abdomen; passing to the rear, yet exhibiting to his men no sign indicative of the grevious nature of his injury, he handed over the leadership to General Yule. Mob law, however, takes little cognizance of eventualities: the Peasant Revolt in 1381 collapsed with the death of Wat Tyler, who was stabbed in the belly by the Lord Mayor Walworth (*Figs.* 154, 155); others have averred that

an Esquire of the King, John Cavandish, alighted from his horse and thrust his sword into Wat Tyler's belly. The rebellion collapsed, and the king, Richard of Bordeaux, styled himself champion of the malcontents; the tax-collector breathed his last within the precincts of St. Bartholomew's Hospital.

Multiple bullet-wounds inflicted by the machine-gun furnish eloquent testimony to the scientific accuracy of modern weapons designed to mangle and mutilate, but plurality of knife and dagger wounds tells a story of Latin blood, fiendish hatred, insanity, or determination on the part of assassin, hired or misguided, to make sure of the victim whom he had pledged himself to kill. The

FIG. 156.—St. Sebastian. Martyred in 288 A.D., by being shot to death with arrows. He was popularly invoked for protection against plague. (From a painting by Andrea Mantegna, 1431–1506, at the Vasa d'Oro, Venice.) (*By kind permission of the Wellcome Historical Medical Museum.*)

belly constitutes a convenient target for miscreants: Julius Cæsar had twenty-three wounds, but it was the wound in the "groin", inflicted by Brutus, which evoked the famous reproach and made Cæsar abandon further resistance; Rizzio, done to death in Holyrood Palace in sight of his Queen, had no less than fifty-six gashes, some of which were doubtless abdominal. The art of the painter has depicted St. Sebastian with many arrow wounds of the abdomen (*Fig.* 156), as well as of almost every part of the body; the martyr did not, however, succumb from these injuries, and was finally clubbed to death by Diocletian's orders.

The murderer in his desire to slay may not select the obvious and easy approach to the abdominal cavity; the rectum of Edward II was the avenue chosen by

coarse and brutal men for the insertion of red-hot spits, and a horn on this occasion was a forerunner of the modern speculum for instrumental introduction (*Fig.* 157). Simon de Montfort was killed at Evesham by a penetrating wound through the buttock or rectum*; his corpse was treated with all the barbarity which is associated with civil war—his head was sent to one destination, his limbs dispatched elsewhere, and his testicles sent as a gift to Maud, the wife of Mortimer.

It is not, of course, the prerogative of the purple or of high estate to endure the agonies of an intestinal injury inflicted through the perineal orifices: a poor woman admitted in labour into a provincial hospital was attended by a practitioner unskilled in his art; employing forceps, he first delivered one eye of the babe, and at the second endeavour removed the child and a coil of the maternal small

FIG. 157.—The murder of Edward II at Berkeley Castle about the Feast of St. Matthew. The hot spit was thrust through into the intestines in order that the murder might be committed without leaving mark of violence upon the body; this was enjoined by Maltravers and Gurney.

intestine. A distinguished obstetrician, who was hurriedly summoned, performed a laparotomy and sutured the rent in the uterus; he then closed the end of each segment of the disrupted bowel, replaced the coils inside the abdomen, and demanded that an intestinal surgeon should be summoned to restore the continuity of the gut. The day of extreme specialization has its disadvantages (!); a certain amazement at the type of operative technique employed by a great obstetric teacher, and natural reluctancy to subject the woman to an immediate second operation, led to disastrous

* Tandem nec vi nec multitudine adversum solum veteranum prævalentis, armis suis a parte posteriori detectis, sicam ejus fundo servulis manus impressit. Sic extractum e sella solotenus alii capite truncant, alii manus abscidunt," etc. (*Chronicon de Lanercost.*)

"Nevertheless neither force nor numbers were successful against such a veteran; his armour being opened at the back, the hand of a foot-soldier thrust in his dagger to the hilt. So having withdrawn it from the buttock, some cut off his head, others tore off his hands," etc.

procrastination, and when the writer finally arrived the patient was moribund from general peritonitis.

More fortunate was the patient to whom Bland-Sutton[4] was summoned early; a practitioner undertook a curettage to relieve dysmenorrhœa; the uterus was perforated by a dilator, and a coil of ileum protruded through the hole. The doctor, thinking that this was a product of conception, withdrew more than six feet of intestine; cutting off one end, he tugged at the other and returned the patient to bed, thinking she would surely die. Bland-Sutton opened the abdomen, closed the rent in the uterus, resected that portion of the mesentery from which the intestine had been separated, anastomosed the ileum to the cæcum (for the distal end of the severed intestine had been torn out of this portion of the large gut), and the patient recovered.

Sarmoff also reported the case of a woman of 36, whose uterus was perforated by a curette and a number of coils of small bowel escaped through the vagina.[5] The abdomen was opened one-and-a-half hours later; subtotal hysterectomy was performed, and despite the ablation of 15 ft. of small intestine the woman got well.

The Old Testament contains several accounts of murderous and indeed treacherous abdominal wounds inflicted by the ancient Hebrews upon unsuspecting foemen and strangers. The mental and moral uplift for the youthful reader of these chapters in the Scriptures which recount the crafty assault of Ehud the Benjamite,* of Joab,† or of Abner,‡ is a matter for conjecture. On the other hand, for those unattracted by the Books of the Kings and Judges, a similar disservice may be performed by the modern cinema, which in exchange for a few paltry coins, daily and nightly, portrays to those of an impressionable age the violence of treacherous and lecherous gangsters; the manifest avidity of mankind to appeal to force must also forcibly suggest to youth that might is right, and that the spear and sword cannot now or ever be turned into ploughshare or pruning hook.

The vilest and the most brutal injuries have been those dealt by perfervid religious enthusiasts in the name of the Church; the Huguenots appear to have rivalled Catholics in the devilry of their tortures: "Master John Jerome of Edam, with some catholic friends of the town of Horne, were captured and taken to a place commonly called Scugen, situated in the north of Holland, where, after having been eight times horribly tortured—from which some of them died—they were bound naked on their backs, and on their bellies were placed basins upside down, with underneath them live rats, and above the basins a fire was made; and by the great heat which resulted the rats were driven to eat their way through the belly, and so to escape into the body: by which inhuman tortures all these innocent souls were given to God" (*Fig.* 158). It is also recorded that "They laid open the belly of a living priest, tore out his entrails, put oats in the empty space and made a manger of it for their horses" (*Fig.* 159).

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* "The hilt went in after the blade, and the fat closed after the blade, for he did not draw the dirk out of the belly." (*Judges* iii. 22, Moffatt.)

† "Then Joab took Amasa's beard in his right hand as if to kiss him, and as Amasa was not on his guard against the sword in Joab's left hand, he stabbed him in the belly and with one mortal blow made his bowels gush out on the ground." (2 *Samuel* xx. 10, Moffatt.)

‡ But when Abner came back to Hebron, Joab took him apart to the side of the town-gate for a quiet talk, and stabbed him there in the belly. So he died." (2 *Samuel* iii, 27, Moffat.)

The Saints in several centuries appear to have been marked down for special abdominal barbarities; St. Sebastian, St. Simeon, St. Erasmus, and many of the Christians in the time of Julian perished in this way.

FIG. 158.—From the *Theatrum Crudelitatum Hæreticorum Nostris Temporis* 1588. (R. Verstegan.)

FIG. 159.—From the *Theatrum Crudelitatum Hæreticorum Nostris Temporis* 1588. (R. Verstegan.)

Save in the Orient, where the belly is traditionally disembowelled by suicides, self-inflicted abdominal injury is not a popular modern method of escape from this life, but those who profess a knowledge of the Old Testament will readily recall how Saul fell upon his sword, as also did Gaius Cassius, Julius Cæsar's murderer,

at Philippi in the last century before Christ. Plutarch[6] tells us that Cato the Younger and Mark Antony both killed themselves by this mode; neither Roman perished immediately from the wound. The manner of the former's death has lasted longer in men's minds than has the record of his life: after reading in the *Phædo* of Plato he opened his belly; a physician, urgently summoned, vainly desired to return within the abdomen the intestine of the recalcitrant Roman. On the other hand, Antony's entrails are hardly likely to have unpleasantly extruded themselves, for he died in the embrace of his famous and fastidious queen.

Abdominal wounds of self-infliction are not all made with suicidal intent: a surgeon's scalpel accidentally slipped from his hand and, gliding off the towels which covered the patient as she lay on the operation-table, produced a deep, yet non-penetrating wound of the abdomen, necessitating exploration.

A woman in the pangs of childbirth, thinking that she was about to die, seized her husband's razor and incised her abdomen and uterus transversely; after withdrawing the dead child she severed the umbilical cord and manually removed the placenta. Guggenberg was summoned and sutured the wound; the woman recovered.[7]

Abdominal injuries inflicted by the miscreant have swayed and shaken international politics: the gunshot wound of the abdomen[8] which killed an Austrian Archduchess at Sarajevo helped to set alight the great European conflagration of 1914, while an assassin's bullet in 1901 penetrated the stomach and pancreas of a famous President* of a powerful transatlantic people.

The great literature of the world for æons of time has dealt with War; nor are there wanting realistic, anatomical descriptions of abdominal injuries inflicted and received by the Homeric heroes, or by the more recent legendaries and personages of Shakespeare, Matthew Arnold, or Sir Walter Scott. Frölich with admirable patience calculated that the wounds recounted in the twin Homeric poems amounted to no less than 147 in number, of which some were abdominal, and that over three-quarters of the injuries proved fatal. Dame Fortune, or rather Pallas Athene, is alleged to have befriended Ulysses before Troy,[9] when he received a non-penetrating wound of the abdomen from Sokos' spear; the abdominal parietes were partly torn away, yet he was able to slay his assailant. The timely arrival of Telamonian Ajax and of warlike Menelaus saved the hero of the *Odyssey* from the Trojan throng who would have killed him. Abdominal wounds inflicted by Odysseus and his son Telemachus were responsible for the deaths of some of the suitors in that massacre in the halls of Ithaca; Eurymachus[10] had his breast pierced by an arrow from Ulysses' powerful bow, which penetrated downward into the liver; Telemachus struck Leocritus,[11] the son of Euenor, in the pit of the stomach with "the bronze spear-point", which went right through.

The story of the Combat in the *Lady of the Lake* can be repeated by heart by almost every Scottish schoolboy, who knows that thrice the Saxon blade drew blood from Roderich Dhu; the further information vouchsafed in the following lines suggests at least one wound in the abdominal area:—

> Three times in closing strife they stood,
> And thrice the Saxon sword drank blood;
> No stinted draught, no scanty tide,
> The gushing flood the tartans dyed.

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* President Wm. McKinley. Shot Sept. 6, 1901.

We can read in Matthew Arnold how Rustum killed Sohrab with a spear—"he dropped his covering shield and the spear pierced his side"; and in Shakespeare's drama, Macbeth "unseamed" Macdonald "from nave to chaps" in the manner of one accustomed to wield the claymore—the backhand of the claymore could easily split a man from clavicle to thigh; many of Mackay's soldiers perished in this way, while endeavouring with their muskets to protect their heads in that fierce, overwhelming rush of Graham of Claverhouse's men at Killiecrankie.

FIG. 160.—Martyrdom of St. Erasmus in 303 A.D., by having his bowels torn out by winding them on to a windlass. He was invoked for the cure of abdominal pain and spasms, such as colic. (From a painting in the Vatican by Nicolas Poussin.) *(By kind permission of the Wellcome Historical Medical Museum.)*

FIG. 161.—St. Mamert, one of the local saints of Brittany, who was invoked for the cure of abdominal pains. He is represented in monkish habit with his cloak open, and supporting in his hands his intestines. *(By kind permission of the Wellcome Historical Medical Museum.)*

Penetrating wounds of the peritoneum are therefore the prerogative of passion; accident usually engenders those injuries to the entrails where no breach of belly-wall exists; yet even in the sphere of accident the abdomen appears to be under the special protection of heaven, or perhaps of that wondrous muscular reflex of its encompassing parietes. The referee and the rules of the National Sporting Club may abolish abdominal injuries due to boxing, but passion knows no 'belt' or transpyloric plane, and on rare occasions the angry blow may damage intestine, and the spleen of a spouse has been ruptured by an olive branch (!) wielded by an irate husband (Mondor).

It has always appeared ironical to me to write or read of the " bowels of compassion ", when the contents of the belly have been exposed to public view after and even before the death of the victim by cruel executioners of Tyburn and elsewhere; when St. Erasmus' intestine was coiled around a windlass by fanatics (*Fig.* 160); when in the coronation of a new king of the Baganda of Central Africa eight helpless captives were clubbed and disembowelled, and their intestines hung round the chamberlain's neck so as to make him strong and faithful in his office of state;[12] and when in China prisoners have been taken to the water-edge and tortured by brutally dragging loops of intestine from their abdomen and leaving the coils still attached at one end to their owner, while the other dependent portion has been offered to the fishes of the sea.

The " bowels of compassion " supported in the hands of St. Mamert, one of the local saints of Brittany, who was invoked for the cure of abdominal pain (*Fig.* 161), may graphically indicate the manner of his martyrdom and death.

The infliction of injury upon the dead has always been regarded with abhorrence, except when men's minds and reason are blinded or warped by the lust of war, and in olden days when crimes such as treason were punishable with frightful barbarities. Achilles maltreated the dead Hector; Homer frowns upon this, and arranges that the Gods themselves miraculously preserve the corpse from all defacement, until it is given worthy burial. The idea of carrying enmity beyond the grave was repugnant to the Greek spirit from very earliest times, and Theseus and Hercules are regarded as having first enjoined that there must be a truce so that the vanquished might bury their dead; a duty to the dead was observed even when warfare was most bitter and the adversary a barbarian. The spear-thrust in the side of Christ may have been the compassionate *coup de grâce* of a more kindly Roman soldier who wished to end the agony on the Cross, or may have been a a further act of a coarse brutality which does not cease with the death of the victim.

Excavations in Essex, and doubtless elsewhere, have revealed skeletons buried at the cross-roads, staked through the belly to the earth that shrouded them; " who does must rue ", and those self-righteous persons responsible for the disposal of the bodies of criminals took elaborate precautions that punishment should be everlasting, and that these malefactors should not arise at the resurrection!

The treatment of penetrating wounds of the bowel, although still fraught with grave anxiety, has advanced since the days of Roderick Random, the creation of Smollett's genius;[13] one of his examiners at Surgeon's Hall, London, had little knowledge and little faith that success would ever attend any form of surgical intervention for intestinal injury. The ignorance of an examiner may be concealed under the cloak of the bully, and it may be recalled that the hero of Smollett's novel, during the early part of his viva voce, had to listen to the jibes of a disgruntled surgeon against Scotsmen " who have overspread us of late as the locusts did Egypt "; that road leading south beyond the Cheviots and the Tweed was an avenue of one-way traffic even then!

The *occasional immunity of viscera* in penetrating wounds of the abdomen can no longer be denied; the belief entertained by Makins and by Cheatle since the South African War that a penetrating wound of the belly might involve no visceral damage, found confirmation in the Great European conflict; patients

presenting through-and-through wounds, the track of which lay seemingly across the intestinal area, occasionally exhibited on laparotomy no lesion of a hollow viscus, but a peritoneum containing a variable quantity of blood from omentum, mesentery, abdominal wall, or a retroperitoneal hæmatoma. More dramatic is the story of the apache who, when endeavouring to escape from justice, was transfixed and pinned to the floor by a bayonet: four days later he was cured (Mondor).

The possibility of *spontaneous recovery from a proved wound of the stomach* was demonstrated by Makins, Cuthbert Wallace, Green-Armytage, by Elliot and Henry and others during the Great European War, and this is also true of the large intestine, especially the ascending and descending colon. Gordon Bell's case of *spontaneous cure of multiple small intestine wounds* is one of the miracles of the European conflagration: a soldier was shot from side to side through the abdomen at Loos in 1915; he was not operated upon, and made an uneventful convalescence. The man was shot again in the belly at the First Battle of the Somme in 1916, and this time submitted to laparotomy; there were several perforations of the small gut in the vicinity of a conglomerate mass of adhesions; the recently-wounded and the adherent segments of bowel were ablated by Bell, and the man again made a good recovery. The specimen exhibited an entero-anastomosis between two adjacent coils, and there were also several small herniæ of the bowel mucous membrane, indicating the spontaneous healing of the previous perforations. A thoughtless orderly committed the specimen to the incinerator!

Year by year from 1915 onwards the results of the military surgery of the abdomen steadily improved; more rapid evacuation of those wounded in the belly to casualty clearing stations or advanced abdominal centres, increasing experience of operators, but especially blood transfusion, played a part in diminishing the mortality. In respect of blood transfusion British surgeons had much to thank their transatlantic confrères; to the late Bruce Robertson, of Toronto, my own indebtedness is heavy. In the beginning of 1917 the following sentence appeared under the superscription of a distinguished surgeon: "of 145 cases of abdominal injury, of gunshot origin, with pulses over 120, only 15 recovered—a mortality of nearly 90 per cent"; while by another it was recommended that operation should be denied to those whose pulse-rate was over 140. These words furnished the incentive and spur to effort; results improved, and even in my own hands a series of abdominal cases exhibiting a pulse-rate of over 120 who were transfused and operated on gave a 41 per cent recovery; and another personal series where the pulse-rate was 140 and over gave a 37·7 per cent rate of recovery.[14] Whereas a series of 151 abdomino-thoracic wounds collected and reported by Sir Anthony Bowlby in 1917 exhibited a recovery-rate of 49 per cent, 207 cases operated upon in 1918 by myself and Fourth Army surgical colleagues yielded a recovery-rate of 66·6 per cent.[15] Some operators, such as Charles Saint, of Cape Town, and the late Jack Anderson, a surgeon *sans peur et sans reproche*, had figures as flattering as 81·8 per cent and 79·2 per cent respectively. Nevertheless, the spirit of the operators might well have been crushed by the formidable mortality to be faced each day; yet the soul of surgery burned with undimmed glow; even when the scale seemed heavily weighted against patient and surgeon, Death did not always win the gamble. One of the most remarkable cases of abdomino-thoracic injury in the European War was that recounted to me by Richard Charles, of Ipswich:

a Canadian officer, badly wounded, was transfused in a dressing-station and then brought down by aeroplane to a casualty clearing hospital; he survived a left-sided abdomino-thoracic operation, involving the removal of the spleen and the suture of two wounds of the stomach (*Fig.* 162). The patient subsequently returned to duty, gained the Victoria Cross, and finally, at the Armistice, led his regiment of Canadians into Cologne.

Not all were granted that opportunity of demonstrating anew the heroic mould in which they were cast: an officer of the Connaught Rangers was operated upon by me behind St. Quentin in the early days of the Fifth Army Retreat; he had a left-sided abdomino-thoracic wound, which necessitated a transdiaphragmatic

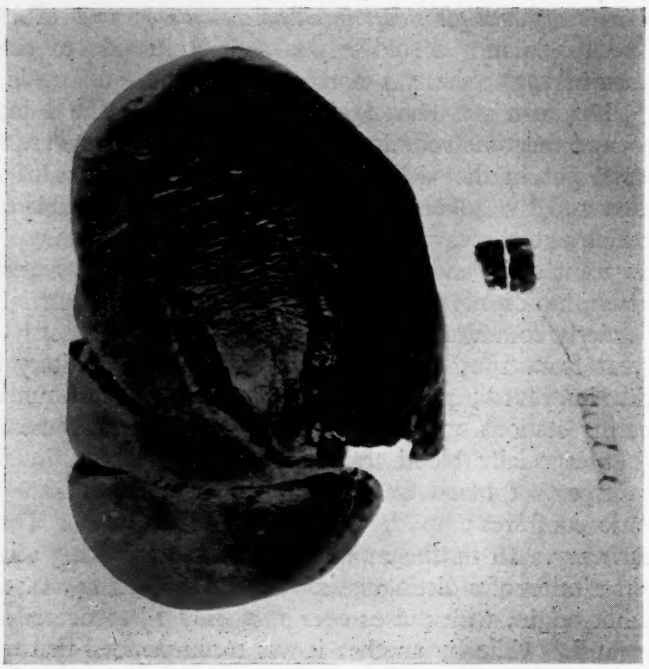

FIG. 162.—Abdomino-thoracic wound of left side; exhibiting ruptured spleen and the piece of high explosive removed. (Mr. Richard Charles's case.)

splenectomy, the closure of two large wounds in his stomach, the repair of the diaphragm, pleural 'toilet', and closure of his chest. Despite the difficulties attending the evacuation of wounded during that period of turmoil and tribulation, he was able subsequently to write from Ireland, thanking me for what I had done, and expressing his eagerness and feverish anxiety to return to France and to rejoin his South Irish Division. *ἀλλ' οἱ γὰρ ἀθυμοῦντες ἄνδρες οὔποτε τρόπαιον ἐστήσαντο.*

The possessive spirit has always characterized such as can display in a drawing-room the products of their own disordered metabolism; biliary and urinary calculi and the vermiform appendix are often preserved by their owners as appropriate exhibits when friends are gathered together. The soldier, not unnaturally, clings to the missile which has been extracted from his body, and cherishes this symbol of his hardihood with a humour that is denied the bereaved

recipient of a posthumous decoration. *Figs.* 163–166 exhibit the missile, the patient, and their reunion after nearly twenty years ! This case has already been recorded in the BRITISH JOURNAL OF SURGERY,[15] and describes a double resection of bowel and many other visceral injuries ; a small abscess round a minute fragment of metal in the depths of the pelvis recently led to his re-admission under my care, when the piece of high-explosive weighing 3 oz. was restored to him.

The conflicting emotions of a certain Scottish nursing sister might well be the theme for novel or play ; this lady, distraught with personal grief over the death of her betrothed, a regimental medical officer to a Guards' battalion who had been killed by enemy gunfire a day or two before, nursed back to life, amongst others, a German prisoner-of-war taken in the later days of the Cambrai Battle. This man, only

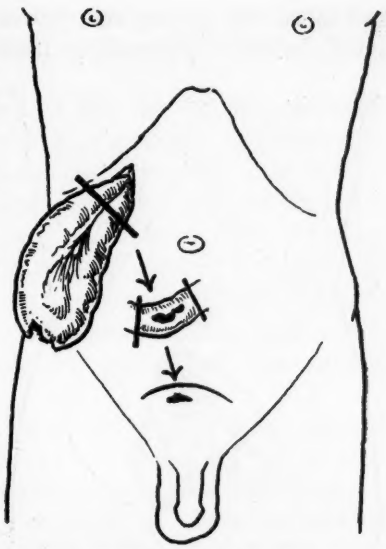

FIG. 163.—Diagram of injuries to bowel and bladder in case described in text : double resection of bowel ; bladder wound sutured.

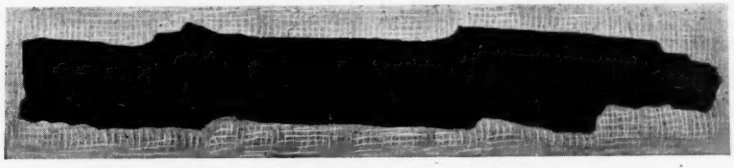

FIG. 164.—Same case. Fragment of high explosive, weighing 3 oz., which produced the injuries to the abdominal wall and viscera, necessitating a double bowel resection.

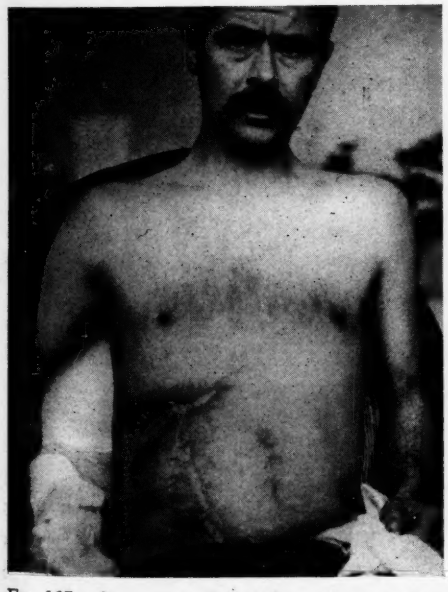

FIG. 165.—Same case. Patient from whom the 3-oz. piece of metal was removed, one year after wounding.

FIG. 166.—Missile restored by writer *18 years later* to patient, who is holding it in his own hand.

half-killed by a bayonet wound of the abdomen, with two damaged coils of small intestine protruding from his belly, survived a double resection of small intestine at my hands—a rare recovery from an injury produced by so deadly a weapon (*Fig.* 167). It may take an Aeschylus or Cicero, a Montaigne, Bacon, Sterne, or Wordsworth, to write adequately of friends and of the philosophy of friendship; it may require the gifts of Robert Burns to eulogize gratitude; but meetings after many years with patients, whose abdominal injuries are depicted in *Figs.* 168, 169, furnish ample attestation that gratitude is not only to be measured by the written, or even the spoken, word, although the appeal of an Australian to the writer to bail him out at a Police Court to which he had been haled for a trifling misdemeanour was doubtless made in a spirit of friendship which had been engendered on the Somme!

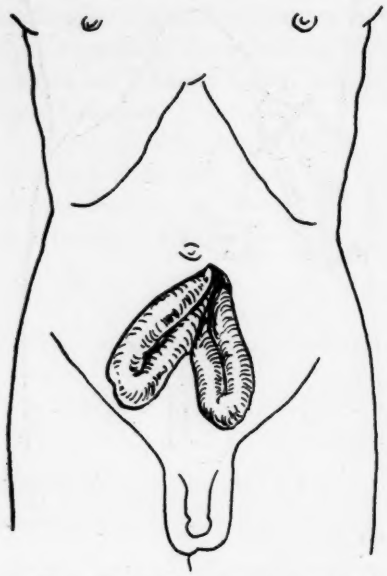

FIG. 167.—Prisoner-of-war; hernia of two coils of shattered bowel. Double resection. Well when seen one month later at base hospital.

Penetrating abdominal injury has on rare occasions added to the sum of human physiological knowledge: the stomach wound of the unfortunate Alexis St. Martin, the Canadian voyageur, was studied with relentless tenacity over many long years by that selfsame Dr. Beaumont who was medical officer to the American troops that captured Fort George in 1812; his careful observations

FIG. 168.—Sergt. B.; bomb wound. Resection of transverse colon and jejunum; suture of sigmoid, wounds of liver, forearm, and left hip. Recovery.

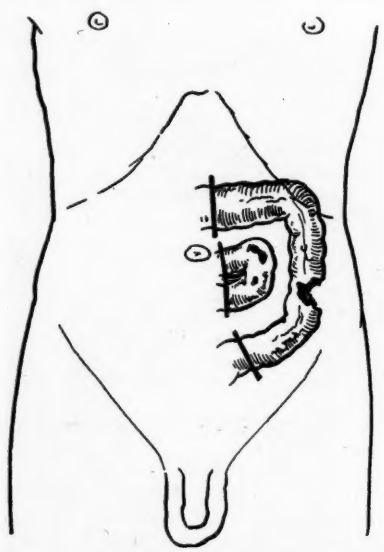

FIG. 169.—L-Corp. Whittaker; penetrating wound of abdomen. Resection of jejunum; resection of distal portion of transverse colon, splenic flexure, and descending colon. Recovery.

constitute the earliest glimmerings of the physiology of gastric secretion, and many of his notes and letters are preserved in the Library of the University of Toronto.

Readers of this address may well be reminded of the title of one of Henry Fielding's papers, " An Essay on Nothing " ; yet it may perhaps be permissible to reiterate that the most frequent cause of abdominal injury is war ; that, despite advances in the operative treatment of such lesions since the days of Roderick Random, surgery is still frequently impotent to save ; and that in those words of commingled comfort, encouragement, and kindly reproof, which Sir Anthony Bowlby would often use to dispirited surgeons, " some men are mortally wounded " ! Military surgery is a carnivorous type of operative technique, offending and revolting to the spirit of artistry that characterizes the modern abdominal interventions of every-day hospital work.

Lord Tweedsmuir, in the concluding pages of his recent work on Augustus,[17] writes, " If his *magna imago* could return to earth . . . he would be amazed by the loud praise of War as a natural and wholesome concomitant of a nation's life " ; those surgeons who have had first-hand experience of abdominal lesions inflicted by the brutal modern methods and missiles of an adversary will share that Augustan amazement.

I have made heavy drafts on the expert knowledge and on the generosity of many friends ; in matters historical no surgeon ever appeals to Sir D'Arcy Power in vain, nor can any librarian surpass Mr. Cecil LeFanu of the Royal College of Surgeons in zeal or avidity to help.

The officials of the Wellcome Historical Medical Museum have been lavish in their generosity, and a number of the illustrations come from that kind source. For *Figs.* 154, 155 I am indebted to the courtesy of the Prime Warden and the Court of Assistants of the Fishmongers Company, who have graciously vouchsafed permission for their reproduction in this paper. To all my most grateful thanks are due.

REFERENCES

[1] *The History of Charles the Great and Orlando*, ascribed to Archbishop Turpin.
[2] DOUGHTY, A. G., and PARMELEE, G. W., *Siege of Quebec*, iii, 190 ; and *Makers of Modern Canada*, ii, 202.
[3] HORACE, *Odes III*, iii, 13.
[4] BLAND-SUTTON, SIR JOHN, *Middlesex Hosp. Arch.*, 1912.
[5] SARMOFF, J., *Ann. of Surg.*, 1923, lxxviii, Dec., 745.
[6] *Plutarch's Lives*, 958, 1148. London : John Lane, The Bodley Head.
[7] HOWKINS, JOHN, *Middlesex Hosp. Jour.*, 1938, Jan.
[8] *The Times*, 1914, June 29.
[9] HOMER, *Iliad*, *XI*, 423 et seq.
[10] HOMER, *Odyssey*, *XXII*, 80 et seq.
[11] *Ibid.*, 298.
[12] FRAZER, SIR JAMES G., *Golden Bough*, pt. iv, ii, 221.
[13] SMOLLETT, TOBIAS, *The Adventures of Roderick Random*, xvii, 108. Hutchinson & Co. : London Classic Novels.
[14] GORDON-TAYLOR, GORDON, *Brit. Med. Jour.*, 1921, Oct. 22, 539 et seq.
[15] GORDON-TAYLOR, GORDON, *Ibid.*, 1919, Aug. 2, 131.
[16] GORDON-TAYLOR, GORDON, *Brit. Jour. Surg.*, 1919, vii, 142.
[17] BUCHAN, JOHN (LORD TWEEDSMUIR), *Augustus*, 1937, 345. London : Hodder & Stoughton.

THE EVOLUTION AND DEVELOPMENT OF SURGICAL INSTRUMENTS

By C. J. S. THOMPSON, M.B.E., PH.D.

HON. CURATOR OF THE HISTORICAL COLLECTION, MUSEUM OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

V. THE VAGINAL DILATOR AND SPECULUM

THE history of the vaginal dilator and speculum, which dates from the early centuries of the Christian era, is one of special interest to obstetricians and gynæcologists. It is more than probable that the idea of the expanding-bladed instrument, which we know from existing specimens was used by the Romans, was evolved from the hand of the obstetrician with the fingers first placed together in the shape of a cone for introduction and then spread outwards for exploration.

This supposition is borne out to some extent by the directions given in Celsus* for examination of the cervix uteri: "The operator", he says, "having greased his hand, ought first to insert the index finger and keep it there until the os uteri be further opened, and then he should again introduce another finger, inserting the remainder in the same way until the whole hand be in the womb."

As the instrument later developed in two distinct forms, viz., the bladed and the tubular, it will be well to consider them separately.

Although Hippocrates makes no special mention of the vaginal dilator, he alludes to two different kinds of rectal dilators similar in construction to the bladed instrument later used for the vagina, and the references to lesions of the cervix uteri lead one to think that some kind of instrument was employed. In the passage on the diseases of women in the Hippocratic treatise, dilators consisting of six flat pieces of pine wood, each being a little longer than the other, are alluded to. These, after being well smeared with oil, were to be introduced with the object of dilating the vagina. (*Fig.* 170, 1.)

The earliest dilators so far known are those of bronze found among the surgical instruments discovered at Pompeii, which was destroyed A.D. 79. One of these has four blades and the other three, and they are worked by different mechanisms. The four-bladed specimen works on a frame, the two upper blades being hinged to the top of the handles and held by a cross-bar tipped with rams' heads. On turning the central screw, the lower blades are drawn outwards, at the same time separating slightly, while the upper blades also diverge. The total length of the instrument is 13 in. (*Fig.* 170, 2.)

The three-bladed dilator, with its central screw mechanism, became the model for later instruments down through the Middle Ages. The blades are prismatic and the points blunted. The two lateral stand at right angles to a flat-sided bar. At their extremities they turn inward and are united to each other by a round hinged-joint. A rod passes through the hinge and is received by a linch-pin on

* A. CORNELIUS CELSUS, Book VII, Cap. 29.

the upper surface. A special feature of the instrument is two flat bars, convex externally and prolonged below the side-bars, each attached to the corresponding side-bar by a hinged-joint. Thus they can be pushed outwards, out of the way, when the screw is being turned, and then pressed inwards so as to meet close above

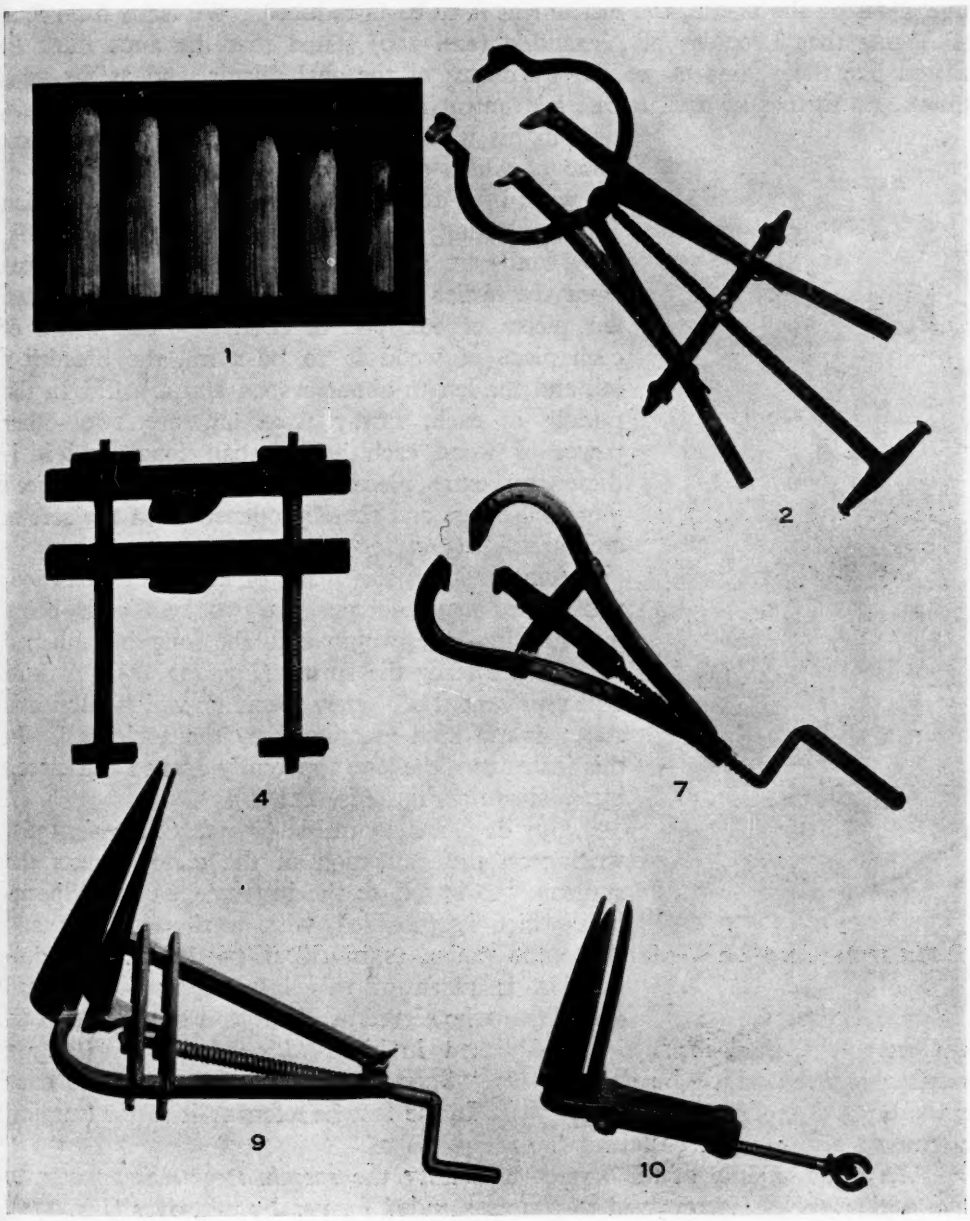

FIG. 170.—1, Flat pieces of pine wood of increasing length, used in the time of Hippocrates to dilate the vagina. 2, Four-bladed bronze dilator discovered at Pompeii (before A.D. 79). 4, Ebony or boxwood dilator described in the earlier of the Albucasis manuscripts in the Bodleian Library (1271). 7, A dilator of the Sixteenth century. 9, A dilator in the Prujean Collection of the Royal College of Physicians. 10, Three-bladed speculum depicted by Scultetus in *Armamentarium Chirurgici* (1672).

the handle of the screw. This arrangement allows the dilator to be firmly grasped. (*Fig.* 171.)

The anatomist and obstetrician, Soranus, who flourished from A.D. 98 to 177, devotes a chapter in his book on gynæcology to the use of the vaginal speculum, beginning with an account of the position to be assumed and describing how, after greasing the blades, the instrument is to be introduced. We learn from Paul of Aegina that Leonidas of Alexandria (A.D. 200) stated that the anus must be dilated like the vagina of women by means of the anal dilator, and as far as is known, no further allusion to the instrument is made until the time of Albucasis, who in his work on surgery describes several types, some of which are depicted in the manuscripts of his work (936–1013). In the MSS. in the Bodleian Library, dating respectively about 1271 and 1465, three kinds are represented. A model of one taken from the earlier MS. has screws at the ends of two flat pieces of boxwood or ebony. "The width of each piece of wood is to be 2 in., the breadth 1 in., and the length about a span and a half. In the middle of each, firmly fixed on, are two other pieces of wood each half a span long, and it is these two extra pieces which are to be introduced into the passage and thereby opened when the screws are turned" (*Fig.* 170, 4).

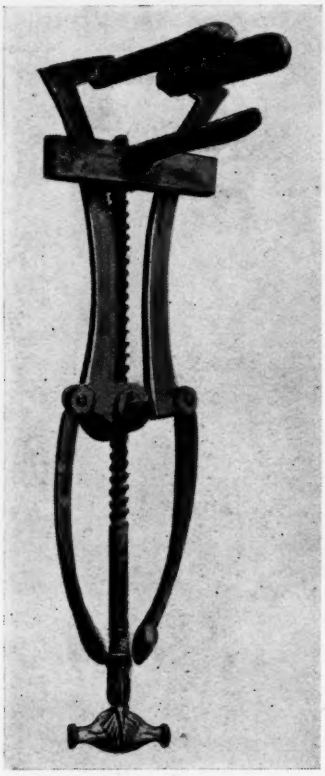

FIG. 171.—Three-bladed bronze dilator discovered at Pompeii (*c.* A.D. 79).

Another kind, also made of ebony or boxwood, was shaped like a forceps, the two bent ends being inserted into the womb and the long bow-shaped handles providing the spring (*Fig.* 172, A). A third type represented is a screw speculum which Albucasis states is "a kind mentioned by the ancients". In this instrument the jaws apparently opened and closed by a single spring (*Fig.* 172, B).

Guy de Chauliac, in his *Grande Chirurgie*, 1363, writing on the extraction of the child, directs the surgeon "to introduce the instrument called Speculum which is provided with a thumb-screw and dilate the vagina as much as possible". No description is given of this instrument, but in 1526 Gersdorff, who served as a surgeon during the wars of Charles the Bold in 1476–77, published in his *Feldtbuchder Wundartzney* a *speculum matricis* which he describes as "Speculum, that is—to open the anus or the birthparts of women" (*Fig.* 173). In the text he refers to it as "a surgical instrument also used in obstetrics".

About the middle of the sixteenth century the *speculum matricis* appears to have excited much interest, and was recommended for use by midwives (*Fig.* 174).

In 1554 Rueff, a surgeon who is said to have superintended the midwives of Zurich, wrote a book entitled *De Conceptu*, in which he figures his *speculum matricis*, which he says is to be used when "children sticking in the womb and being dead are to be brought forth". He gives a long account of how the midwife should

use such instruments, one of which he refers to as the "*apertorium*", a long cross-bladed dilator or the opening instrument, and the other as the "*speculum matricis*" (*Fig.* 175), the "looking-glass of the matrix". There is some evidence that a mirror was used in connection with this instrument.

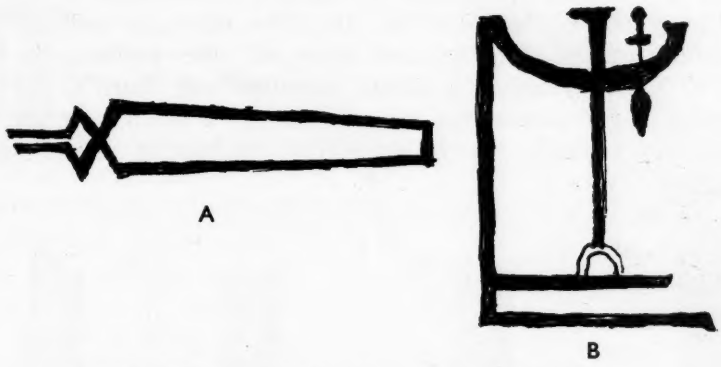

FIG. 172.—Other types of dilator described by Albucasis (1271).

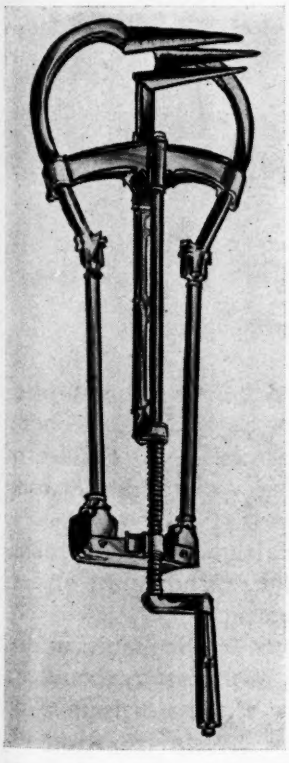

FIG. 173. — The *speculum matricis* of Gersdorff (1526).

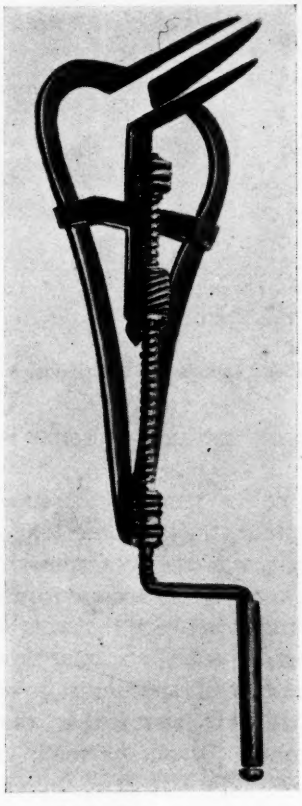

FIG. 174.—A speculum of the sixteenth century.

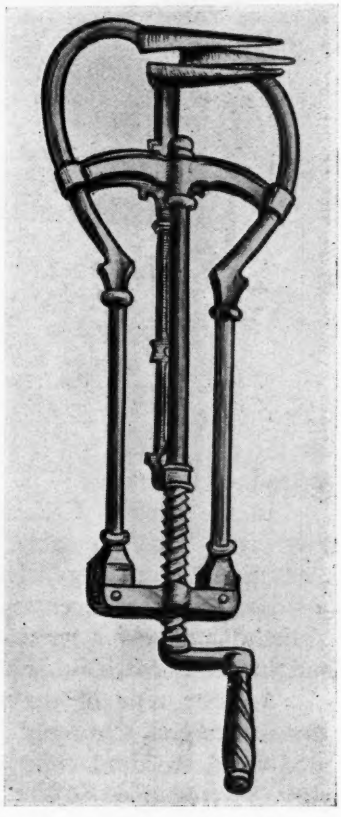

FIG. 175.—The *speculum matricis* described by Rueff (1554).

There can be no doubt that the *speculum matricis* was not meant to be a dilator of the cervix but an expander of the vagina, a speculum in fact as that word is now understood. It is interesting to note that both in Gersdorff's and Rueff's instruments the blades are concave internally and not prismatic.

Later on in the century the vaginal dilator began to undergo a definite development, which is shown in the instruments figured in the works of Ambroise Paré in 1579. In one, termed the *Vielle* type, the screw apparatus and bars are encased up to the blades between plates on their upper and lower surfaces, the screw being worked by a winch. Another is almost identical with Rueff's, the male-screw passing through the horizontal bar and not through a bearing on that bar. This is also worked by a winch. In the third type, the bearing on the horizontal bar

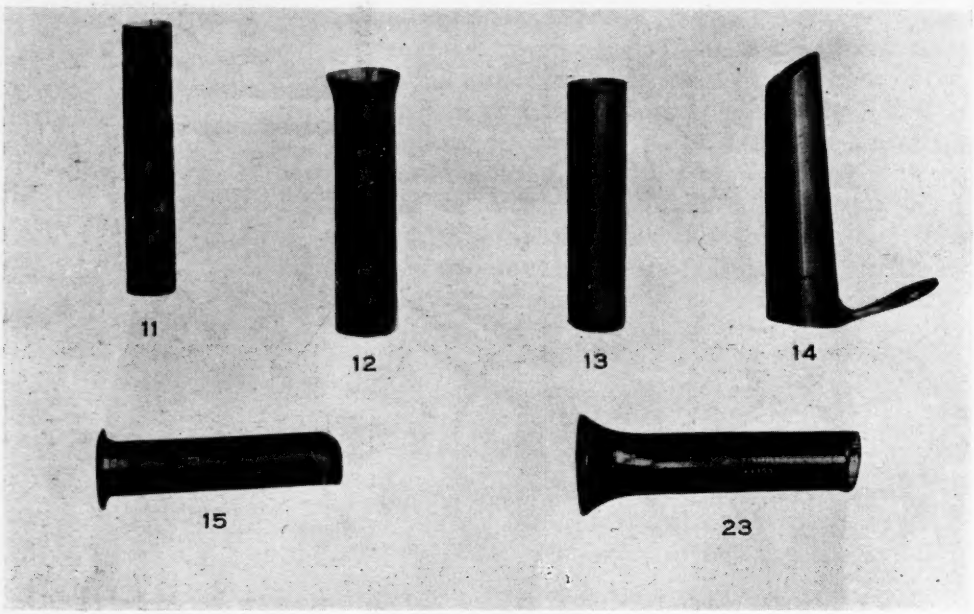

FIG. 176.—Tubular specula (for description, *see* text).

for the male screw is a large metal nut and the screw is worked by a thumb-piece instead of a winch.

In the Prujean Collection of instruments preserved in the Royal College of Physicians is a formidable instrument with three long, sharp-pointed blades each measuring 6½ in. long, which are worked by a central screw (*Fig.* 170, 9).

Early in the seventeenth century Fabricius Aquapendente figured a three-bladed dilator with a screw apparatus between the handles for expanding the blades, and in 1606 Fabricius Hildanus depicted a similar instrument.

Another type of the three-bladed speculum is shown by Scultetus in his *Armamentarium Chirurgici* (1672). He states that the instrument " should be used when the dead child is to be cut out or some ulcer of the matrix is to be viewed " (*Fig.* 170, 10).

Coming to the eighteenth century, we find that marked changes took place in the form of the dilator and speculum and that various modifications of the tubular type came into favour.

The tubular speculum, a gynæcological rather than an obstetrical instrument, was probably of equal antiquity with the bladed type, as the Talmud explains how

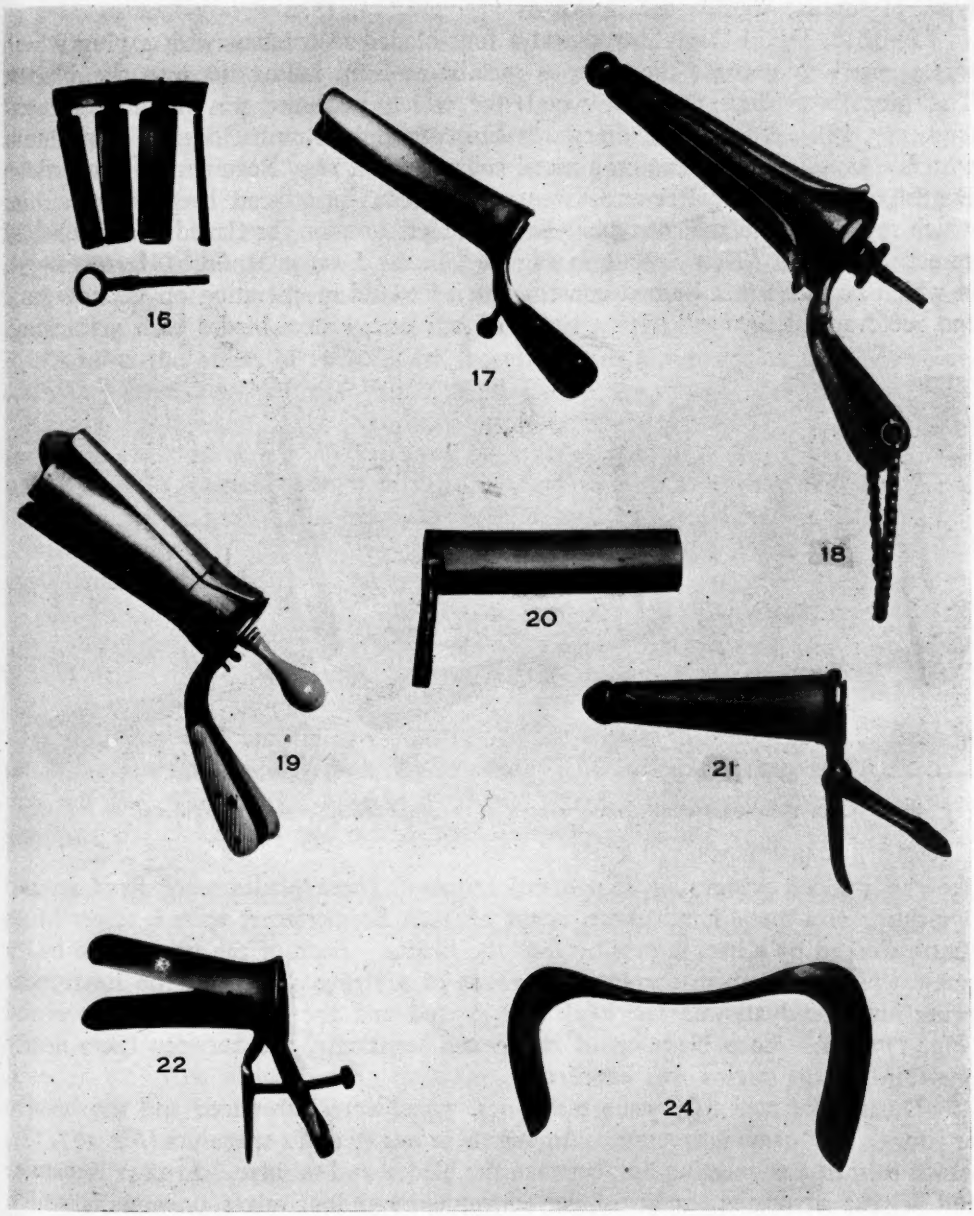

FIG. 177.—Various types of specula. 16, Beaumont's (1837); 17, Ricord's; 18, David Davis's (1830); 19, Charriere's; 20, Recamier and Segalas's (1842); 21, Rizzoli's; 22, Cusco's; 24, Marion Sims's.

a woman may ascertain if blood issues from the womb by introducing a tube into the vagina and passing a rod with a cotton tampon along the tube. The tube may have been a bamboo internode or the stem of a gourd (*Fig.* 176, 11). Metal tubes

were used from an early period, and are mentioned by Mar Samuel about A.D. 160. Although the cylindrical shape (*Figs.* 176, 12, 13) was slightly altered by division into valves from time to time, the tubular remained until the nineteenth century. Later types of tubular specula are shown in *Fig.* 176, 14, 15.

In 1830, David Davis introduced a four-bladed instrument with a plug which was devised to prevent the mucous membrane from falling between the blades. The plugs were first made of wood, for which vulcanite was later substituted (*Fig.* 177, 18). A few years afterwards Magenty and Columbat devised a speculum with five or six short blades on a metal collar, and in 1837 Beaumont, an ingenious English surgeon who afterwards went to Canada, introduced his new speculum which reverted to an ancient type. Before he left London for Canada, he published an account of his "New Speculum Vaginæ" in the *London Medical Gazette* (1837), in which he states that he had constructed it "to aid in operating on vesicovaginal and rectovaginal fistulæ." It consisted of four or five steel blades each 3 in. long,

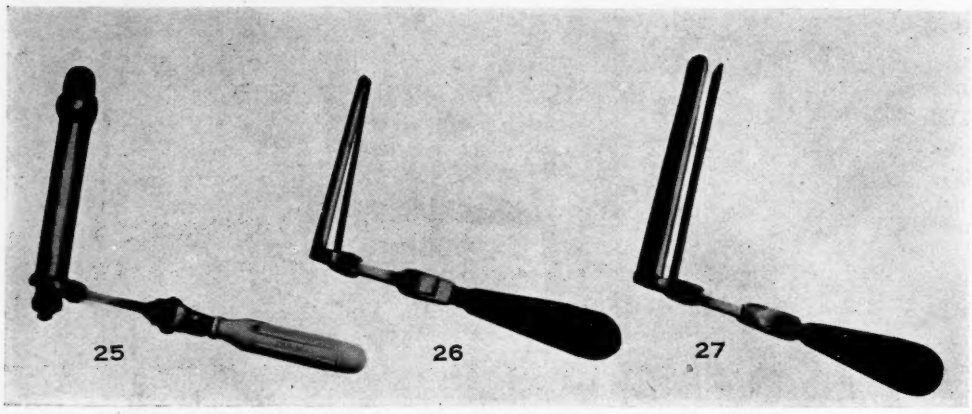

FIG. 178.—Nineteenth-century models of the three-bladed dilator. 25, Plum's 26 and 27, Weiss's.

like the wooden wedges of Hippocratic times. These blades were fixed around two-thirds of a metal hemisphere, about an inch in diameter, with a screw in its centre worked by a handle passing into the blades. Each of the blades also had a screw which drew them together by means of a string, which on the instrument being introduced allowed the blades to expand and the handle to be unscrewed (*Fig.* 177, 16). Each blade could be opened separately, and between them nearly one-third of the vagina was exposed.

During the next fifty years many new types were introduced and the bivalve or 'duck's bill' came into vogue. Among these was Ricord's speculum (*Fig.* 177, 17), which formed a connecting link between the bladed and tubular. In 1842 Recamier and Segalas advocated a split tubular instrument with four valves, only one of which functioned (*Fig.* 177, 20); and in 1753 Heister described a bivalve speculum "to dilate the anus or vagina". Rizzoli, Charriere, Cusco, and Trélat also introduced instruments designed on similar lines (*Fig.* 177, 19, 21, 22), and later, Brambilla, in 1782, figured a two-valved speculum, the long blades of which were operated by a transverse bar with a screw in the centre, and expanded when pressure was put on the handles.

In the early nineteenth century two new instruments were introduced which may be regarded as novelties. The first was a vaginal speculum with glass blades; and later came Palfrey's (Blackbee's) skeleton-bladed instrument, so devised to give an extensive view of the vagina and cervix uteri.

Later on Plum and Weiss revived the use of the three-bladed dilator with three prong-like blades and a plug. It was operated by a screw in the handle, and was recommnded for both uterine and rectal purposes (*Fig.* 178, 25–27).

About 1855, Robert Ferguson (1799–1865), the first Professor of Obstetric Medicine to King's College, began to use a straight tubular glass speculum. In 1870, Sir William Fergusson refers to this instrument as "the speculum that I devised" and it is the type which survives to-day. These instruments were originally made of metal and then of glass mirrored inside, the outside being coated with several layers of gum-elastic. Walsh added a linen web, woven over the first coating of gum in order to prevent breakage (*Fig.* 176, 23).

And so the origin of the tubular speculum with silvered glass inside is somewhat uncertain, but evidence points to Robert Ferguson as the inventor.

In 1845, Marion Sims introduced his double-ended duck-bill speculum, the shape of which he states in *The Story of My Life*, he discovered by chance. He says that when journeying to visit a patient at a distance one day, finding he had forgotten his speculum, he stopped at a hardware store and bought a long pewter gravy spoon. By bending each end backwards at right angles, he improvised an instrument that he found answered the purpose excellently.

Later on, he used a single-bladed instrument made more concave than the spoon bowl, but eventually added a second blade at the end, as he found when one blade was introduced into the vagina the other made an excellent handle (*Fig.* 177, 24).

From this brief outline of the history of the vaginal dilator it will be seen how the tubular outlived the tri-valved *speculum matricis* of the early centuries, and survived the many types of tri-valved and quadri-valved gynæcological instruments of the eighteenth and nineteenth centuries.

Many of the specula illustrated have been photographed from actual specimens in the Historical Collection of the Museum of the Royal College of Surgeons.

VISIT OF THE ACADÉMIE DE CHIRURGIE DE FRANCE TO THE ROYAL COLLEGE OF SURGEONS OF ENGLAND (July 4 and 5, 1938)

THIS is the third meeting since 1936, when, by the happy inspiration of the French Academy of Surgery, these alternating annual visits have been arranged.

Sir Cuthbert Wallace, as President, welcomed our French colleagues in the lecture theatre of the College at Lincoln's Inn Fields at 10 a.m. on Monday, July 4. After a short cinema lecture on the operative treatment of cardiac ischæmia by Mr. Laurence O'Shaughnessy, Professor John Beattie took the visitors round

FIG 179.—The bell which was presented to the Académie de Chirurgie.

the new Bernard Baron laboratories, and various methods of research were demonstrated by the staff. A motor coach took the party by a circuitous route to St. Thomas's Hospital, where lunch was provided. From 2 p.m. till 3.30 various operations (including gynæcological and orthopædic) were performed by Mr. Max Page and other members of the staff. There was also a demonstration of clinical cases and of pathological specimens.

In the evening there was a banquet in the library of the College of Surgeons, where the French surgeons and their ladies, together with other distinguished visitors, were entertained by the Council of the College and their ladies.

At this happy gathering, where French and English languages were equally used, Sir Cuthbert Wallace made a presentation of a silver bell to the Académie de Chirurgie for use by the latter at their meetings. The bell, made and designed by Omar Ramsden, bears the inscription: "Académie de Chirurgie de France—Royal College of Surgeons of England" (*Fig.* 179). Professor Maurice Chevassu made an eloquent reply in returning thanks.

On Tuesday, July 5, the programme began by the party being received at the British Post-Graduate School at Hammersmith by the Dean, Colonel Proctor, and the Surgical Director, Professor Grey Turner. In the tour of the laboratories and wards the latter was ably assisted by his son, who translated fluently into French. After seeing the School and witnessing some operations, Professor Grey Turner fascinated his audience by a lecture, profusely illustrated by pictures, films, and specimens, reviewing various subjects of surgical interest.

After lunch a visit was paid to the Brompton Chest Hospital, when operations were performed by Mr. J. Roberts and Mr. Tudor Edwards.

It is hoped that this happy interchange of friendship and surgical experiences will go on each year and serve to renew the comradeship of twenty years ago.

The following members of the Académie de Chirurgie attended the meeting:—
Professor Maurice Chevassu, *President.*
Professor Raymond Gregoire, *Vice-President.*
Dr. Louis Bazy, *Secretary.*
Professors and Doctors: Henri Hartmann, Pierre Duval, Leon Berard, Oscar Lambret, de Martel, Basset, Picot, Cotte, Fourmestraux, Madier, Petit-Dutaillis, Seneque, Iemtel, Nandrot, Courty, Caravan, Bourde, Perdoux, Leclerc, Blondin, Fruchaud, Ferey, Foucault, Fontaine, Michel-Bechet, Bourde.

TRANSSPHENOIDAL DECOMPRESSION FOR PITUITARY ADENOMA*

By GILBERT PHILLIPS, Sydney

The following three cases are reported as it is considered that they illustrate the merits of transsphenoidal decompression in the surgical treatment of adenomas of the pituitary gland. The present writer has occasionally found it a difficult problem to decide whether a pituitary adenoma should be attacked surgically by the transfrontal route or through the sphenoidal sinus. The former procedure, although it appears at the present time to be fashionable both in England and America, is nevertheless a formidable one and involves a number of risks, any of which, however deliberately the operation may be planned and however delicately it may be executed, may determine a fatal result. Both methods of approach offer equally good opportunities for removing a considerable portion of an intrasellar neoplasm. Eisenhardt[1] has described 2 cases of pituitary adenoma, from Cushing's series, which survived twenty-four years after subtotal removal of the growth. Both these operations were performed by the transsphenoidal route. The transsphenoidal operation is certainly a simpler surgical procedure and does not carry with it the risks attendant on excessive retraction of the frontal lobes, which may be necessary in posteriorly situated growths, and on post-operative diencephalic overaction, which constitutes one of the most formidable post-operative complications.

Post-operative hyperthermia seldom comes as a surprise when it follows surgical attack in the region of the tuber cinereum. Some degree of post-operative hyperthermia is the rule after transfrontal operation if pre-operative evidence exists that the tumour is compressing the floor of the third ventricle. How much this complication depends upon relief of the pressure of the tumour on the heat-regulating mechanism in the diencephalon and how much it is due to traction and retraction by the operator, may probably be estimated by comparing the incidence of this complication in transsphenoidal and transfrontal operations. It is significant in the three cases of transsphenoidal decompression reported here, in all of which there was evidence that the tumour was of a large size and consequently would be expected to the exerting some pressure on the diencephalon, that convalescence in each case was not complicated by an unusual rise of temperature.

In addition to post-operative hyperthermia, cases have been observed, particularly after prolonged operation in people of over 40 years of age, in which transient or persistent mental symptoms have succeeded the removal of the tumour. These symptoms may not be severe, but in some cases have been sufficient to incapacitate the patient. Sudden death may occur in some cases, in the early stages of the transfrontal operation as retraction of the frontal lobe is being carried out. The following summary abstract is an example of such a catastrophe :—

On May 31, 1934, J. N., a young man aged 19, was admitted to Lewisham Private Hospital with the diagnosis of chromophobe adenoma of the pituitary gland. Operation

* From the Department of Surgery, Sydney University.

was performed on June 2 under intratracheal nitrous oxide and gas anæsthesia after induction with ether. After turning down a frontal bone-flap on the left side, the frontal lobe was being retracted from the orbital plate of the frontal bone when the patient's condition, which until that time had been excellent, suddenly depreciated and respiration ceased. The heart stopped one minute later, and in spite of all efforts the patient could not be revived. Unfortunately, permission for an autopsy could not be obtained.

It is not only such risks which may render the transfrontal operation a serious procedure; in some cases unexpected complications may arise although the neurosurgeon has been well satisfied with the technical result of the operation and with the patient's early convalescence. The following case illustrates a catastrophe of this kind :—

On September 30, 1937, D. S., a male aged 49, was admitted to Lewisham Hospital with the diagnosis of chromophobe adenoma of the pituitary gland. This diagnosis was confirmed after investigation in hospital, and on October 15 a transfrontal operation was performed after

FIG. 180.—Post-mortem specimen from the patient D.S., from the anterior-inferior aspect. The neoplasm has been removed and the posterior portion passing downwards to the pons can be clearly seen. The anterior portion of the capsule is distended with fresh blood-clot.

anæsthesia had been obtained by a basal dose of avertin followed by intratracheal nitrous oxide and oxygen. The tumour was exposed after elevation of the left frontal lobe and incision of the dura mater along the lesser wing of the sphenoid bone. Arachnoid adhesions surrounding the tumour were divided with endothermy and the whole of the contents of the pituitary fossa was cleaned out with pituitary rongeurs and a strong sucker. The chiasm was post-fixed so that it was not necessary to section the left optic nerve. There was no oozing in the region of the tumour bed, but a gutta-percha drain was led down into the pituitary fossa and brought out through the angle of the scalp wound. The bone and scalp flaps were then replaced. The operation took four and a half hours, a blood transfusion of 500 c.c. being given half an hour before the conclusion of the operation. During the next half an hour the patient became conscious and responded rationally to questions. He was returned to the ward in good condition. Twelve hours after operation his temperature rose to 102° F., but on sponging with 70 per cent alcohol it fell to 98°.

On the morning after operation his condition was excellent, he was perfectly rational, well oriented, and co-operated well during examination. His temperature was 99°. The first dressing was done two days after operation, when the drainage tube was gently removed, following which a few cubic centimetres of serosanguinous fluid escaped from the wound. On the afternoon of October 17 the temperature rose to 102·5°, and during the next two days a hyperthermia persisted at this level, broken only by falls of temperature following sponging with 70 per cent alcohol and intramuscular injections of acetylsalicylic acid. On October 20 his temperature fell to normal. He felt extremely well, his mental condition was quite lucid, his appetite was good, and micturition was taking place normally. At 8.30 p.m. on October 21 he suddenly became unconscious and died three-quarters of an hour later.

At post-mortem examination on October 22 a recent extensive hæmorrhage was found extending over the left frontal region, apparently arising from the base of the brain. On elevating the left frontal lobe the pituitary fossa was found to be full of recent blood-clot, but no tumour substance. On section of the optic nerve and retraction of the chiasm, it was found that the posterior clinoid processes and the dorsum sellæ had been eroded, and that at least two-thirds of the bulk of the original tumour was lying in the posterior fossa between the cerebral peduncles and the basisphenoid. The secondary hæmorrhage had taken place into the tumour tissue which had been left behind, its capsule being distended with receral clot (*Fig.* 180), and had spread from there around the left frontal lobe on to the superolatent surface.

In other cases a remote complication may sometimes appear which may not at first be associated causally with diencephalic trauma. Cushing[2] has reported 5 cases in which hæmorrhagic erosions of the gastric or œsophageal mucous membrane had taken place following operations for intracranial tumour. All these cases were fatal, and in three of them the cause of death was peritonitis succeeding acute perforation. Cushing concluded that the cause of hæmorrhagic erosions and necrosis, with hæmatemesis and often followed by acute perforation, was a neurogenic one, and was produced by autodigestion of mucous membrane which had become necrotic following intense vasoconstriction as the result of excessive discharge from autonomic centres in the diencephalon. The following case, which fortunately was not fatal, illustrates a complication of this type following an attack on a pituitary adenoma by the transfrontal route. It is to be noted that the structures in the pituitary region were bound together by arachnoid adhesions, during the separation of which considerable traction of the floor of the third ventricle was necessary.

On September 22, 1936, Mrs. E. D., aged 53, was admitted to Lewisham Hospital with a diagnosis of chromophobe adenoma of the pituitary gland. Operation by the transfrontal route was performed on September 29, and after retraction of the left frontal lobe the optic nerves and chiasm were exposed. A small pituitary tumour was seen projecting just in front of the anterior border of the optic chiasm, and the tumour, chiasm, and optic nerves appeared to be bound together by fine arachnoid adhesions. Some considerable time was spent separating these adhesions, and a subtotal removal of the tumour was made by pituitary rongeurs without section of the left optic nerve. At this time the operation had been proceeding three and a quarter hours and the anæsthetist reported that the pulse was irregular in volume and rhythm. The blood-pressure was 125 systolic, 90 diastolic. During the closure the pulse continued to be irregular and the anæsthetist considered that auricular fibrillation was present. Just before the operation was completed the patient was given a 350 c.c. blood transfusion, and after one hour's rest on the operating table was returned to the ward in better condition but still unconscious. An hour later she vomited three ounces of clear fluid, and during the succeeding three hours she again vomited, on three occasions, small quantities of fluid which were progressively more blood-stained. She was conscious and responding six hours after operation, and her general condition was good. She continued in this manner until midnight on Oct. 1 (54 hours after operation), when she suddenly vomited 300 c.c. of

blood. This hæmatemesis was succeeded by three others, each of the same volume, at about half-hourly intervals. It was estimated that in all she had lost about two pints of blood. A slow, continuous blood transfusion was commenced, and allowed to run for about two hours. In all, 1000 c.c. of blood were given. Her condition was very unsatisfactory, she was quite unconscious, her temperature had risen to 102°, and although the pulse, which had been imperceptible before the transfusion, had improved, the rate was 150 and it was most irregular. The blood-pressure was 95/45 and from time to time she displayed Cheyne-Stokes respiration. Her condition did not improve very much during the next two days, and it was thought that she might still be bleeding into the bowel. With this possibility in mind, another blood transfusion was given on Oct. 4. At 10 p.m. on Oct. 4 she passed two pints of old blood per rectum. Two more blood transfusions were given, one on Oct. 5 and one on Oct. 6, between which, at 10 a.m. on Oct. 6, she passed another two pints of old blood per rectum. Her condition, however, had been gradually improving over the last two days and she was now quite conscious and co-operative. The remainder of her convalescence was relatively uneventful, except for one or two episodes of mental obfuscation. She was discharged quite well on Oct. 31.

The following case reports of transsphenoidal pituitary decompression illustrate the simplicity of the surgical technique, the absence of post-operative complications, and the satisfactory result to the patient. In these three cases operation was

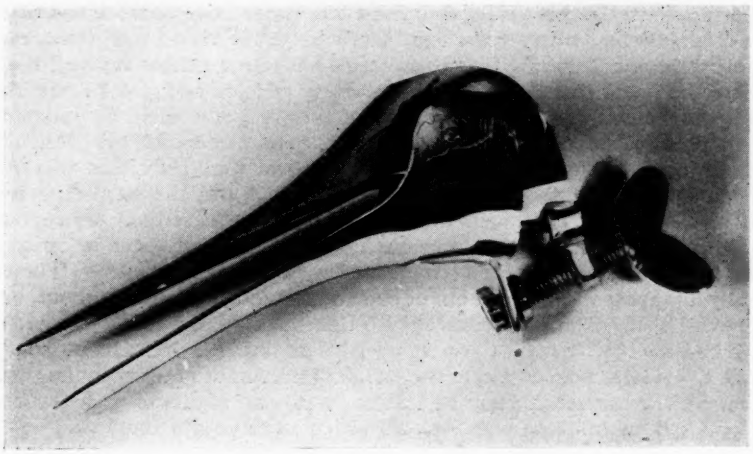

FIG. 181.—Duckbill self-retaining retractor used for transsphenoidal approach through one nostril. (Modification by Mr. Richard Flynn, M.S., F.R.A.C.S.)

performed through one nostril, using a self-retaining retractor (*Fig.* 181). This approach was found to provide an adequate exposure of the tumour without the necessity of any incisions being placed at the junction of the nose and the cheek or beneath the upper lip. The early stages of the operations were carried out in each case by an otorhinolaryngologist, who, after submucous resection of the cartilaginous septum, then removed the vomer and the inferior and posterior parts of the perpendicular plane of the ethmoid bone, thus exposing the crista sphenoidalis, which serves as a guide to the midline. The duckbill self-retaining retractor was then inserted and opened as widely as possible, displacing the mucous membrane over the conchæ sphenoidales until the antero-lateral apertures of the sphenoidal sinus were seen. This is an important point, and ensures an adequate exposure of the anterior wall of the sphenoidal sinus. The anterior wall was then removed and the mucous membrane lining the sinus drawn out. When this was done the

sella turcica was exposed and was found in each case to be eroded in several places. The remainder of the sella turcica was then removed with punch forceps and the tumour exposed. The tumour may be gently separated from the walls of the pituitary fossa by passing the tip of a small glass sucker tube around its periphery, and after this manœuvre it can be grasped with pituitary rongeurs and drawn down into the sphenoidal sinus. During this procedure a gush of cerebrospinal fluid makes its appearance, and continues to drain freely. The question as to whether much of the tumour should be removed or not will be discussed later. The nose may be plugged with gauze for twenty-four hours. The cerebrospinal rhinorrhœa usually ceases on the third day of convalescence.

CASE REPORTS

Case 1.—Mr. J. C., aged 53.

HISTORY.—The case was referred to Lewisham Hospital on May 26, 1934, by Dr. Conrad Blakemore, who had detected an incongruous bitemporal hemianopia. The patient was an intelligent co-operative man, an accountant by profession, who stated that he had been a healthy man all his life, and had had no operations and no serious illnesses. There was no history of any neurological disease in his family. The symptoms of his present illness had appeared insidiously and his chief complaint was progressive depreciation of vision. He began to wear glasses first thirty years ago, not because his vision was deteriorating at that time but because he was suffering from headaches. Wearing glasses relieved the headaches. Nine years later he found that his sight was becoming affected and that he might miss seeing a letter or a figure which was placed at the beginning or end of a line. He tolerated this state of affairs until three years ago when he began to suffer from severe bilateral frontal headaches, which usually appeared before rising in the morning and were associated with nausea. On advice from an ophthalmologist his glasses were changed and the headaches more or less disappeared, but his vision continued to depreciate and he described his central vision as 'smoky'. During the last twelve months he has been awakened once or twice a week by severe frontal headaches, which were accompanied by a feeling of nausea. These headaches would generally disappear when he got out of bed and commenced to move about, but occasionally they persisted all day. He has noticed also that the temporal field of vision in each eye has definitely become restricted in the past twelve months. He has had no actual vomiting and no epistaxis. He has noticed a considerable depreciation of potency in the past five years. He stated that for the last seven years he had practically no nights of undisturbed sleep, but that his sleep has been troubled with dreams which were always more or less of the same character. They would commence with a sense of futility, and then he would dream that he was back in the past and that the dream was familiar to him. In his dream he would be expecting trouble all the time and would be glad to wake up.

ON EXAMINATION.—The following positive findings were made :—

1. Rather prognathous jaw; teeth show some separation. The skin is silky and has a waxy pallor and the hair of the head is very fine. There is considerable subcutaneous fat which, however, has the normal male distribution. The hands are large and the fingers spatulate. Radiographically the terminal phalanges show definite tufting.

2. Fundi show temporal pallor of both optic discs.

3. The visual acuity is 6/24 in the left eye and 6/18 in the right eye. The visual fields show a bitemporal hemianopia with the upper quadrants chiefly involved (*Fig.* 182).

4. The direct sellar ray shows considerable erosion of the sella turcica and of the posterior clinoid processes (*Fig.* 183).

5. The basal metabolic rate is −21 per cent.

DIAGNOSIS.—Pituitary adenoma (probably mixed chromophile and chromophobe).

OPERATION.—On May 29 an attempt was made to turn down a frontal flap to reach the pituitary neoplasm. Owing to the extreme vascularity of the scalp the operation was discontined during the cutting of the bone-flap, as the patient was becoming exsanguinated. On June 15 a transsphenoidal approach was made through one nostril. After exposure and removal of the conchæ sphenoidales and the lining membrane of the sphenoidal sinus, the

eroded sella turcica was exposed, and was removed with punch forceps and a blunt hook. A large, smooth, plum-coloured tumour was thus exposed, and a small portion removed with pituitary rongeurs for histological examination. The tumour was then grasped with pituitary rongeurs and slowly drawn down into the sphenoidal sinus. During this procedure copious

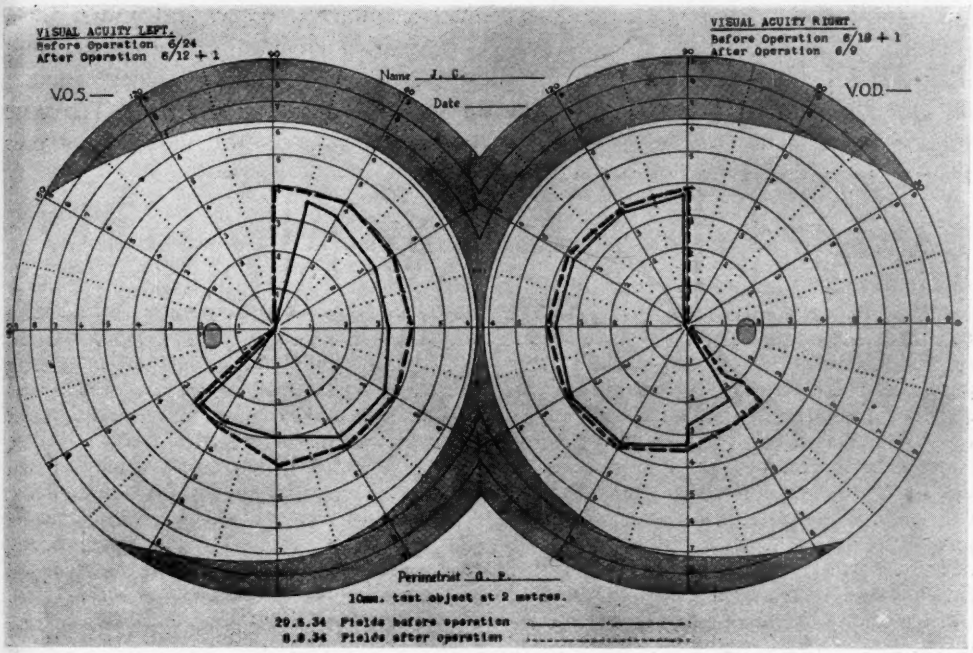

FIG. 182.—*Case* 1. Visual field charts, showing bitemporal hemianopia, mainly involving the upper quadrants. As can be seen, there was slight extension of vision after operation.

FIG. 183.—*Case* 1. Radiograph showing erosion of the sella turcica and the posterior clinoid processes.

drainage of cerebrospinal fluid took place. The pituitary needle was then passed into the substance of the tumour, but no cystic fluid was detected. Convalescence was quite uneventful. After operation the temperature did not rise above 99.4°, and was never above normal after the second day. The cerebrospinal rhinorrhœa gradually diminished, and ceased on the fourth day. There was no polyuria or polydipsia following operation. He was discharged from hospital ten days after operation.

SUBSEQUENT PROGRESS.—When interviewed seven weeks after operation, the patient said he felt very well and had had no headaches since operation. Since his discharge from hospital he had slept soundly at night and had had no dreams. Subjectively his vision had improved considerably. On examination it was found that the visual acuity had improved to 6/12 + 1 in the left eye and 6/9 in the right eye, although the extent of his fields had increased only very slightly (*Fig.* 182).

The patient was interviewed again recently—that is, three and a half years after operation. He has been working consistently during the whole post-operative period and his visual acuity is still 6/12 in the left eye and 6/9 in the right eye. He is delighted with the fact that his visual acuity permits him to do all his own book-keeping. He has been quite free from all symptoms and has succeeded in expanding his business considerably in the past two years.

HISTOLOGICAL DIAGNOSIS.—Mixed adenoma (chromophile and chromophobe).

Case 2.—Mrs. E. L., aged 51,

HISTORY.—The patient was referred by Dr. Holmes à Court on Dec. 8, 1937, as a case of pituitary adenoma. Her present illness commenced eleven years ago. Previous to that time she had always been a healthy woman, had had no operations, and had never consulted a doctor. In 1927 she had an attack of severe bilateral frontal headache lasting for about a week. The headache used to come on at night and last throughout the greater part of the day. It was not accompanied by nausea or vomiting. At the end of this week she had a sudden loss of all vision, which was accompanied by relief of headache. This blindness persisted for one month, after which she had a gradual return of vision over the succeeding three months. The sight in the left eye completely recovered, while the right eye she states was not at that time " as good as the left ". Since then she has suffered from occasional bilateral frontal headaches, usually coming on in the early morning and generally not very severe and not accompanied by nausea. The menopause took place ten years ago, at the age of 41, and for the preceeding three years the menses had been extremely irregular. She has been gradually losing weight and has lost about 4 st. in the past eight years. There has been no polyuria or polydipsia, and no change in the appearance of the face or the size of the feet or hands. She very often becomes drowsy in the day-time, and has fallen asleep on several occasions while seated at her midday meal.

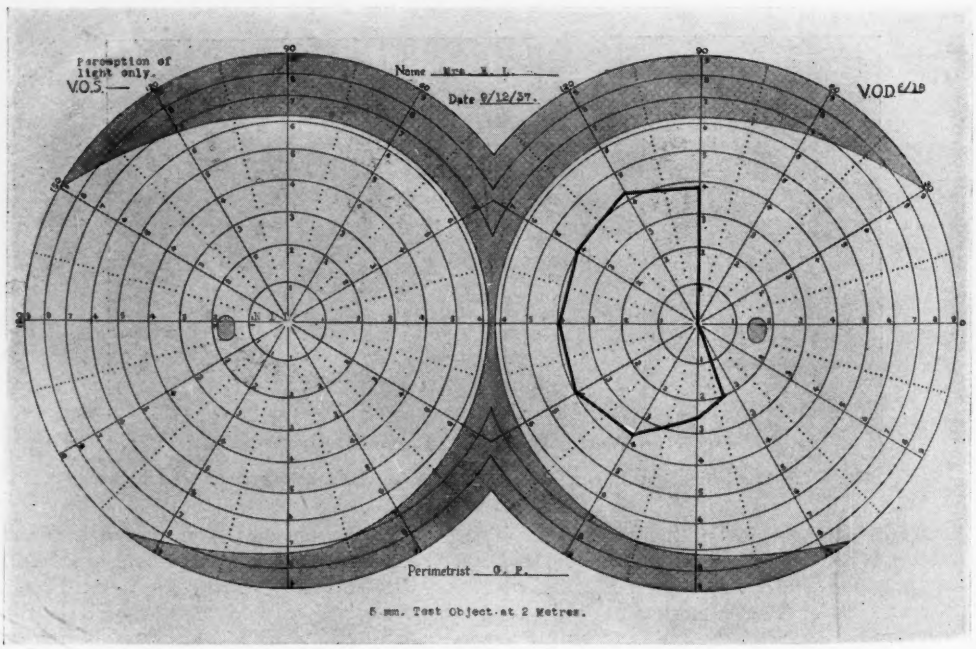

FIG. 184.—*Case* 2. Visual field charts, showing temporal hemianopia in the right eye.

Two weeks ago she had an attack of diplopia, which lasted for one day and was succeeded immediately by considerable nausea and frontal headache. On the next day she noticed that her eyesight was not as good as usual, and from that time up to the present she thinks her eyesight has depreciated further, particularly in the left eye.

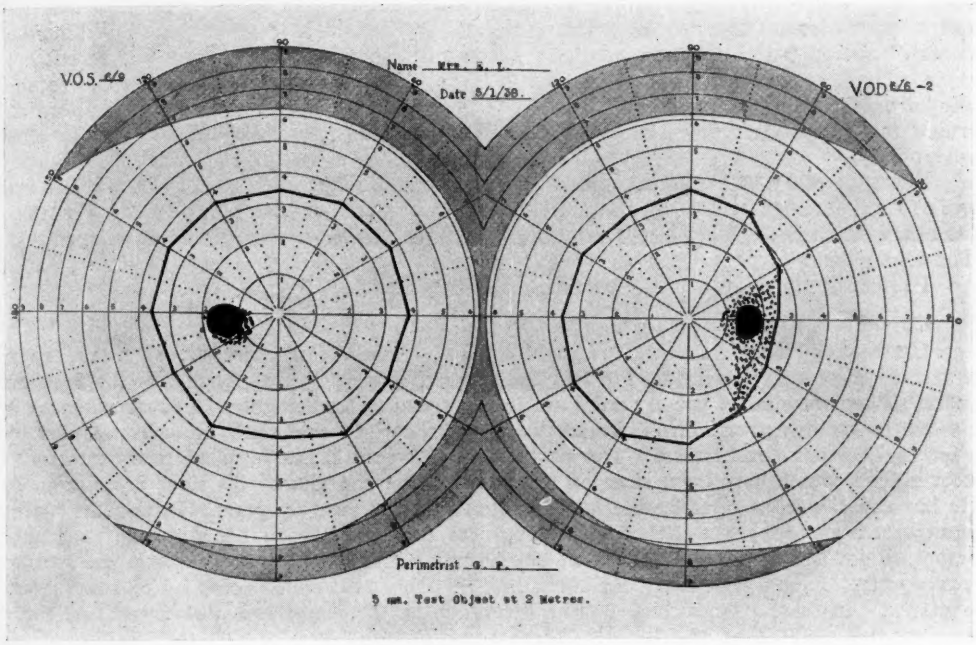

FIG. 185.—*Case* 2. Showing the marked improvement of vision in both eyes after operation.

FIG. 186.—*Case* 2. Radiograph showing erosion of the sella turcica and the posterior clinoid processes. The postero-superior outline of the tumour can be visualized.

ON EXAMINATION (Dec. 9).—Primary optic atrophy was detected in each fundus. Visual acuity in the right eye was 6/18 and in the left eye she had perception of light only. Examination of the visual field in the right eye disclosed a temporal hemianopia (*Fig.* 184). A direct

sellar radiograph showed marked erosion of the sella turcica, and one could almost visualize the outline of the neoplasm (*Fig.* 186). The basal metabolic rate was −14 per cent.

DIAGNOSIS.—Chromophobe adenoma of the pituitary gland.

OPERATION.—On Dec. 14 a transsphenoidal operation was performed, and after removal of the eroded sella turcica a plum-coloured neoplasm was disclosed; this was gently drawn down into the sphenoidal sinus with pituitary rongeurs. No cystic fluid was detected and a small piece of the neoplasm was removed for histological examination. Within four hours of operation the patient was able to distinguish objects in the room with the left eye alone. Her temperature did not rise above 100° F. and was normal from the second day onwards. Her convalescence was quite uneventful. The cerebrospinal rhinorrhœa ceased three days after operation, and she was discharged from hospital two weeks after operation.

SUBSEQUENT PROGRESS.—On examination on Jan. 5, 1938, visual acuity in the left eye was 6/9 and in the right eye 6/6, and there was marked return of vision in both fields (*Fig.* 185). On examination on Feb. 3 she felt extremely well and had had no symptoms since operation. The visual acuity and the visual fields were the same as at the previous examination.

HISTOLOGICAL DIAGNOSIS.—Chromophobe adenoma.

Case 3.—Mr. W. A., aged 46.

HISTORY.—The patient was referred by Dr. Layton Spearman on Nov. 14, 1937, as a case of pituitary tumour. He had been suffering from frontal headaches for fourteen years. These headaches usually come on in the morning and might also occur when he is excited or unduly worried. They are worse on the right side, and are accompanied by a feeling of malaise and nausea. During the past five years it has been necessary for him to wear glasses, which have been changed recently. Using the glasses his sight is quite good. He has experienced a considerable loss of potency in the past six years. In the last twelve months he has had three attacks of unconsciousness, each one taking place in the early morning before he got out of bed. In these attacks he was unable to be roused and would breathe stertorously. Each attack lasted for about half an hour, and was followed by a rather severe headache. In these attacks there was no incontinence, the tongue was not bitten, and there were no movements of the limbs. There were no gustatory or olfactory hallucinations preceding these attacks. He has noted during the last two years that his head has increased slightly in size. He has had to take the band out of his hats to make them somewhat larger. There was no history of polyuria or polydipsia, and there has been no recent change in weight.

ON EXAMINATION (Nov. 20).—He appeared to be a well-nourished man, the skin of whose face looked coarse and felt thick. His nose was very large and prominent and the lips full and almost negroid in appearance. There was no obvious prognathism of the jaw and no separation of the teeth. The hair of the head was very coarse and plentiful. The normal male distribution of fat was present. His hands were large and the fingers tended to be spatulate. Further examination of his nervous system disclosed no abnormal physical signs. The basal metabolic rate was − 18 per cent. Glucose tolerance test disclosed no abnormality. X-ray examination of the skull showed marked erosion of the sella turcica and of the posterior clinoid processes (*Fig.* 187). Visual field examination showed an early upper bitemporal quadrantopsia (*Fig.* 188).

DIAGNOSIS.—Mixed adenoma of the pituitary (chromophile and chromophobe).

OPERATION.—On Nov. 20 a transsphenoidal approach was made to the pituitary gland through one nostril. After removal of the anterior wall and the lining membrane of the sphenoidal sinus, it was found that the central portion of the sella turcica was completely eroded and that the neoplasm had commenced to decompress itself into the sphenoidal sinus. After removal of the lateral portions of the sella turcica the pituitary needle was passed with difficulty into the neoplasm, which was extremely tough and rubbery. No cystic fluid was detected. An attempt to grasp the neoplasm and draw it down into the sphenoidal sinus was made, but the tumour appeared to be adherent above, and as blood-stained cerebrospinal fluid was draining freely, this manœuvre was abandoned in case intracranial hæmorrhage might be started. The temperature rose to 100° F. twelve hours after operation, but gradually subsided to normal on the second day. The cerebrospinal rhinorrhœa ceased on the fourth day. He was perfectly well and ready to be discharged from hospital ten days after operation when he caught a severe cold and was kept in hospital until Dec. 12.

SUBSEQUENT PROGRESS.—Visual field examination on Jan. 5, 1938, revealed that the upper bitemporal quadrantopsia had disappeared (*Fig.* 188). He stated that he thought his vision had improved. He had no headaches since operation and was eating and sleeping well.

FIG. 187.—*Case* 3. Radiograph showing erosion of the sella turcica and the posterior clinoid processes.

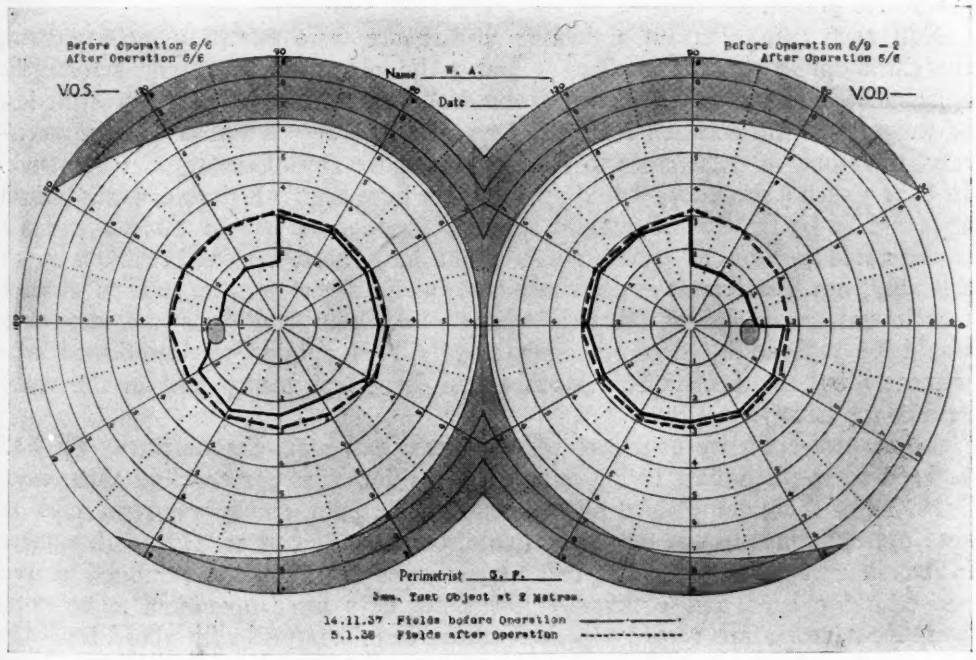

FIG. 188.—*Case* 3. Visual field charts, showing early upper bitemporal quadrantopsia and its disappearance after operation.

During further questioning he admitted that before operation he had a continual feeling of depression and lack of confidence in himself which he stated had now completely disappeared. At a further interview on Feb. 8 he stated that he was feeling quite well and had returned to his work as a tobacco-blender two weeks previously.

CASE COMMENTARY

These three cases present several features in common. In all of them symptoms had been present for several years and the neoplasm was of considerable size, judging both from the presence of neighbourhood symptoms and signs and from the appearances at operation. In each case there had been considerable erosion of the sella turcica, and in *Case* 3 this erosion had proceeded so far that the centre of the tumour was found bulging through the eroded floor of the sella turcica. Although chiasmal pressure was slight in *Case* 3, this may have been due to a prefixed chiasm and a posteriorly situated tumour, since the posterior clinoid processes were practically entirely eroded and signs of diencephalic disturbance were present. The attacks of early morning unconsciousness in this case are difficult to explain. From the absence of an homonymous field defect, olfactory or gustatory hallucinations, and mental or emotional states characteristic of temporal lobe invasion, it would appear unlikely that these episodes were due to cortical involvement by the growth. Dr. A. K. McIntyre, of this Department, has suggested that these may have been attacks of spontaneous hypoglycæmia, as he has seen similar attacks in another patient in the absence of any disturbance of glucose tolerance, which passed off either spontaneously or after administration of glucose. This explanation appears to be not unlikely, since it was known in *Case* 3 that the diencephalon was affected and the attacks did take place in the early morning post-absorptive period.

In each case, signs of endocrine disturbance were relatively inconspicuous, the chromophile element in *Cases* 1 and 3 did not produce a frank acromegalic appearance, and the only evidence of metabolic disturbance in *Case* 2 was the loss of weight and the occasional diurnal hypersomnia. It was not considered necessary, therefore, in these cases to attempt to remove any of the tumour substance. In *Case* 3 such a procedure would probably not have been practicable, as the growth appeared to be tightly adherent to the structures above it. In *Cases* 1 and 2 a considerable portion of each growth could have been removed without much difficulty, but the tumour in each case came so far down into the sphenoidal sinus that it was considered at the time that all neighbourhood signs and symptoms would be relieved by decompression alone. This opinion was confirmed subsequently by the remarkable improvement in visual acuity and in the visual field of the left eye in *Case* 2.

The post-operative course of these patients was most encouraging. They all suffered some discomfort owing to persistent rhinorrhœa for the first three days, but none of them complained of any considerable pain, and it was remarkable to note the difference in the post-operative appearance of one of them with another patient in the next bed who had had a transfrontal operation and exhibited marked peri-orbital œdema and ecchymosis for some days after operation. The post-operative temperature records in these three cases contrast with those following the transfrontal operation, where more or less hyperthermia is not unusual. There was no troublesome polyuria or polydipsia after the transsphenoidal approach, and no signs of mental disturbance.

CONCLUSION

The chief practical consideration in the treatment of pituitary adenoma is relief of the neighbourhood symptoms and signs resulting either from chiasmal

or diencephalic pressure. It would seem undesirable, when this can be produced satisfactorily by a simple surgical procedure which involves no risk other than the slight one of ascending infection during the period of cerebrospinal rhinorrhœa, to embark upon a major, time-consuming operation, the result of which must remain uncertain for some time after operation, and which may involve section of one optic nerve or trauma of the frontal lobe. More particularly is this so since the transsphenoidal approach not only does not interfere with a subsequent transfrontal operation, but by relief of diencephalic pressure diminishes the post-operative risks of the latter procedure if it should become necessary as the result of subsequent visual deterioration.

I have referred to the risk of meningitis following transsphenoidal decompression as a slight one, and I believe that such is the case, provided the operation is carried out between the flaps of mucous membrane on either side of the nasal septum, vomer, and perpendicular lamina of the ethmoid. This field, in the absence of sphenoidal infection, is reasonably sterile, and provided that the patient is nursed in the Fowler position after operation to ensure dependent drainage of cerebrospinal fluid, there does not appear to be any reason why the risk of infection should be other than inconsiderable.

In conclusion, a plea is made for more earnest consideration of the transsphenoidal approach to the pituitary gland when it is certain that the growth is intrasellar in origin and when the chief aim of the operation is the relief of neighbourhood symptoms.

I am indebted to Dr. W. A. Dunn and Dr. Robert Paterson, who performed the sphenoidal exposure in *Case* 1 and *Cases* 2 and 3 respectively.

REFERENCES

[1] EISENHARDT, LOUISE, "Long Postoperative Survivals in Cases of Intracranial Tumor," *Proc. Assoc. Res. in Nerv. and Ment. Dis.*, 1935, xvi, Dec.

[2] CUSHING, HARVEY, "Peptic Ulcers and the Interbrain", Balfour Lecture, 1931; *Pituitary Body and Hypothalamus*, 1932, 177 (Baltimore, Md.: Charles C. Thomas).

AN UNUSUAL CASE OF PROGRESSIVE STREPTOCOCCAL ULCERATION

By H. H. LANGSTON

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THE following case of streptococcal infection presents features of such unusual type that the history appears worth recording :—

HISTORY.—

May 20, 1934.—R. W., a healthy boy aged 10 years, attended his private doctor complaining of pain in the left axilla. On examination soft, tender, discrete glands were palpable in the axilla. Further examination revealed an almost healed septic focus on the middle finger of the left hand, said to have been of a fortnight's duration. The axillary adenitis was treated by hot fomentations, but two days later the child developed a temperature of 104° F., a rapid pulse, and a scarlatiniform rash. An injection of 10 c.c. of concentrated streptococcal serum was given, and with this the temperature and pulse returned to normal, but the swelling in the axilla became fluctuant and was opened for drainage of pus. The wound so made failed to heal, and became a chronic sinus with considerable daily discharge which showed no signs of ceasing.

September, 1934.—Enlarged glands were palpated in the region of the clavicle.

November, 1934.—The boy was admitted to one of the London hospitals, a diagnosis of tuberculous axillary adenitis having been made.

November 12, 1934.—A block resection of the axillary glands was carried out. The operation was easy, and, " as all the infected tissue appeared to have been removed, the wound was closed without drainage ".

A few days later, however, the sutures began to cut out ; on removal, the wound broke down completely, and an active ulceration of the edges of the wound, which spread rapidly into the surrounding skin, commenced. This was accompanied by a copious, purulent discharge.

The glands removed were reported on by the Clinical Research Association as follows : " The glands show a well-developed granulomatous hyperplasia with suppuration into several centres. The change is in no way specific, but a simple, chronic suppurative one. There is no evidence of malignancy."

No further special investigation to establish the diagnosis of tuberculous infection was made, but in February, 1935, the boy was admitted to the Lord Mayor Treloar Hospital, being sent in as a case of " tuberculous ulceration of the axilla, secondary to tuberculous axillary glands ".

ON ADMISSION (Feb. 12, 1935).—Patient pale, thin, and cachectic.

Local Condition.—Left axilla : There was a large, irregular, ulcerated area involving the whole axilla and extending on to the anterior surface of the anterior fold and down the medial aspect of the arm for two inches. The surface of the ulcer was covered by unhealthy granulation tissue. The skin edges were discoloured, irregular, and undermined. In the centre of the ulcer was a sinus which extended upwards towards the apex of the axilla for one and three-quarter inches. *Left shoulder* : Elevated by the spasm of the trapezius. The arm was held in a position of 40° abduction, and was fixed in this position by muscle spasm, very little movement being possible. There was no enlargement of the cervical, right axillary, or inguinal glands, no liver enlargement, and no albuminuria. A radiograph of the left shoulder-joint showed general rarefaction, but no bony erosion.

The blood Wassermann reaction was negative.

PROGRESS OF THE DISEASE.—

Feb. 28, 1935.—Light strapping extension was applied to the upper arm in order to increase the abduction of the shoulder-joint and improve the access to the ulcerated area.

March 6, 1935.—A hæmorrhage of moderate degree occurred. Morphia was given and the extension removed. The hæmorrhage ceased.

March 11, 1935.—A large hæmorrhage occurred, and blood was seen spurting from the sinus opening. The wound was packed, and morphia and rectal saline were given.

March 12, 1935.—The child's condition was obviously very poor. The wound was still oozing slightly, and all the signs of a severe degree of anæmia were present. It was therefore decided to ligate the third part of the subclavian artery. A small transfusion (200 c.c.) was given first and the artery ligated in the classical manner. The operation proved somewhat difficult owing to spasm of the trapezius muscle elevating the clavicle. Following the operation, the radial pulse disappeared and the hand was cold and blue for about three hours, after which the circulation improved, and the hand became pink.

BLOOD-COUNT :—

Red blood-corpuscles	1,375,000
White blood-corpuscles	12,100
Hb	24 per cent

Differential White-cell Count :—

Polymorphs	75·0 per cent
Eosinophils	1·1 per cent
Lymphocytes	18·7 per cent
Monocytes	5·2 per cent

A further transfusion was given.

March 14, 1935 (two days after the above operation).—It was reported that culture of a specimen of pus from the wound revealed the presence of a virulent strain of *B. diphtheriæ* and of a hæmolytic streptococcus. It was therefore thought, at first, that possibly the *B. diphtheriæ* was the causal organism, and 56,000 units of diphtheria antitoxin were given subcutaneously, without any local effect. Meanwhile the hæmolytic streptococcus was subcultured and isolated in pure culture. It was then found that injection of this organism alone into the subcutaneous tissue of the abdominal wall of a guinea-pig produced necrosis, ulceration, and finally sloughing of a large area of the abdominal wall. This occurred in every guinea-pig into which this particular subculture was injected. Later subcultures were less virulent, and strains of hæmolytic streptococci isolated from the pus from the ulcer three months later failed altogether to produce this effect. On no occasion were tubercle bacilli isolated from the lesion, either by culture or guinea-pig innoculation, although several attempts were made to do so.

In view of these reports, the diagnosis of the condition was revised to streptococcal ulceration of the axilla, the *B. diphtheriæ* being regarded as a secondary infection, virulent to a guinea-pig, but non-toxic to the patient.

For a time after the ligation of the artery, the ulcer appeared cleaner and the skin edges in places showed signs of growing in. This was accompanied by improvement in the child's general condition. After a time, however, the condition became stationary and remained so for from two to three months, in spite of various forms of local and general treatment. An autogenous streptococcal vaccine was administered, local and general light treatments were given, bland oily dressings, saline dressings, antiseptic dressings, etc.—all were tried without effect, or, at the most, transient improvement.

July, 1935.—The boy began to complain of shooting pains down his arm and the ulcer began again to extend.

Early August.—The boy again had a large hæmorrhage (five months after the first), but apparently venous in character. From this he very nearly died. A transfusion was given as soon as possible, but bleeding immediately recommenced. The ulcer was therefore packed tightly under gas and oxygen anæsthesia, and a further transfusion given.

Next day under general anæsthesia the axilla was explored, and it was found that the sinus led into a large cavity in the apex of the axilla and deep to the pectoral muscles. The muscles were divided and a large septic cavity exposed; across the apex a bare nerve-trunk was exposed, and, deep to it and surrounded by clot, was found the axillary vein. The nerve-trunk was divided as high up as possible and the axillary vein ligated above and below. It was impossible to separate the vein cleanly from surrounding structures, and in ligating it other portions of the brachial plexus were damaged.

After this operation no further hæmorrhage occurred, but the arm was very largely paralysed, all movement in the forearm and hand being absent, and sensation lost in the areas supplied by the seventh and eighth cervical and first thoracic nerves.

Again for a time the ulcer appeared to improve, and for some months it appeared as if the ulcer would slowly heal.

Early in 1936.—Progress ceased.

March, 1936.—The wound edges were excised by diathermic cautery and the whole ulcer surface cauterized. Again improvement, and at first rapid healing followed, only once again gradually to cease.

Towards the end of May, 1936.—The ulcer suddenly again began to spread rapidly on the arm; the process seemed to be spreading along the fascial planes, so that new sinuses appeared some distance from the original ulcer. These always appeared in a characteristic fashion—as a spot of purplish discoloration of the skin, becoming indurated and then breaking down and becoming ulcers with a dirty grey-yellow base and purplish undermined edges.

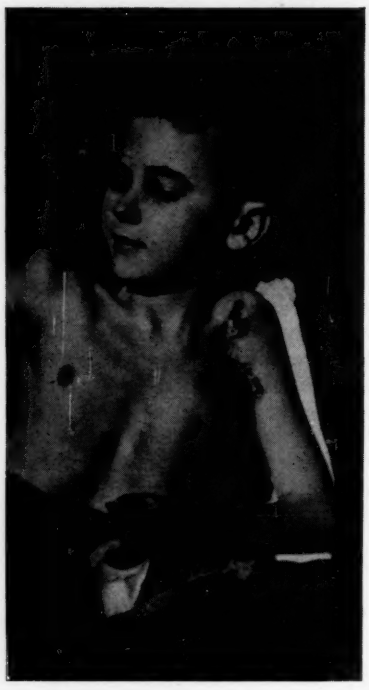

FIG. 189.—Photograph of patient, showing extent of ulceration and complete paralysis of arm.

By this time the ulcer was very extensive, extending down the arm two-thirds of the way to the elbow, up to the clavicle above, on to the chest to within one inch of the nipple, and round the posterior axillary fold behind to the border of the scapula. The skin edges were extensively undermined, purplish-blue in colour, and markedly inrolled. The paralysis of the limb was now almost complete (*Fig.* 189).

May 28, 1936.—The arm was disarticulated at the shoulder-joint. The operation proved easier than was anticipated, as the axillary contents consisted almost entirely of grey necrotic granulation tissue in which no normal structure could be identified. Very few vessels were encountered. The unhealthy skin edges were widely excised; no attempt was made to close the wound, which was swabbed with pure carbolic and dressed with acriflavine emulsion.

The boy stood the operation well and it was now possible to dress the area effectively and keep the extensive ulcer present clean. In spite of this, healing was still very slow: the skin edges in places appeared to be growing in satisfactorily, but in others to be absolutely stationary, and, furthermore, where the ulcer involved the chest wall, it was feared that in time the ulceration might spread through intercostal spaces to the pleura.

November, 1936.—An attempt was made to accelerate healing by skin-grafting with Thiersch and pinch grafts. The attempt was a total failure; the grafts not only would not take, but were apparently rapidly digested.

About this time, in searching for some effective form of treatment, our attention was directed towards an article by Meleney[1], of New York, entitled "Zinc Peroxide in the treatment of Micro-aerophylic and Anaerobic Infections".

In this article Meleney describes 6 cases of progressive ulceration due to hæmolytic streptococcus and their successful treatment by zinc peroxide after a multitude of treatments by other methods had all been unsuccessful.

In his cases, the infecting organism was either a micro-aerophylic or definitely anaerobic hæmolytic streptococcus (4 cases), or at least a hæmolytic streptococcus that would grow slightly better anaerobically than aerobically (2 cases). This feature had led him to search for a slowly dissociating peroxide as an agent which would provide an 'atmosphere' as unfavourable as possible for the growth of an anaerobic organism. Of the available non-toxic peroxides, zinc peroxide seemed eminently suitable, being easily and cheaply obtained and dissociating into the commonly used zinc oxide, in itself a suitable dressing for the ulcer.

Although the case here reported does not appear to have been due to a streptococcus that was even a facultative anaerobe, the description Meleney gives of his 6 cases of progressive ulceration due to a micro-aerophylic hæmolytic streptococcus is clinically so strikingly similar to the case here described that it appeared possible that this might be a similar lesion, and that it might respond to the same treatment.

From November, 1934, onwards.—The ulcer was dressed with a preparation of zinc peroxide in an olive-oil base (the ointment so made thickly covering the ulcer, and being carefully introduced under all undermined skin edges with a probe). The improvement in the condition was dramatic. In a few weeks skin edges that for months had been discoloured, undermined, and entirely stationary, were showing signs of active growth on to the ulcerated area.

April, 1937.—The whole of the large ulcerated area, at one time measuring 8 by 7 in., was entirely skinned over.

Since then (one year) there has been no recurrence, and the boy is leading an active normal life at a preparatory school.

DISCUSSION

This case appears to merit report on account of the unusual character of the lesion present: unusual in its resistance and relentlessly progressive nature, and unusual as a lesion caused by a hæmolytic streptococcus.

No similar case appears to have been reported in the British literature, although the 6 cases reported by Meleney previously referred to appear clinically identical.

Meleney's cases occurred chiefly in relation to inguinal gland and pelvic infections, but he describes the lesion as commencing around a sinus which fails to heal in the ordinary way. He describes the spread of the ulcer—spreading in connective tissue and undermining the skin; spreading in fascial planes and appearing at the surface again at a site some little distance from the original ulcer—which extends in a manner exactly similar to the present case. He mentions the absence of gangrene and the relative absence of tenderness, also characteristic of this case.

The condition is obviously not the same condition as that termed " progressive post-operative gangrene of the skin ", fully described by Stewart Wallace[2] in 1933 and by others since. In this latter condition, as its name implies, there is actual gangrene of areas of skin and superficial tissue. The condition progresses more rapidly, the skin edges are raised, œdematous, and exquisititely tender, and the infecting organisms are usually a non-hæmolytic streptococcus, generally micro-aerophylic, and a specific staphylococcus, the two organisms probably acting symbiotically. Meleney,[3] in an earlier article, sharply differentiates between the two conditions.

The marked improvement, after many months of disappointment, following the introduction of treatment by zinc peroxide, merits especial emphasis. It is possible there are other cases of progressive ulceration in which similar treatment would prove effective. It would seem, too, that it is possible that such cases may be wrongly labelled as due to tuberculous ulceration, and that in such cases careful bacteriological examination with both aerobic and anaerobic culture of material obtained and guinea-pig innoculation might throw an unexpected light on the condition.

SUMMARY

1. An unusual case of progressive ulceration of the axilla involving successively the axillary artery and vein and the brachial plexus is described.

2. The isolation of a streptococcus with abnormal powers of producing spreading ulceration is recorded.

3. Successful treatment by zinc peroxide after many other forms of treatment had been employed is described.

4. The possibility is suggested that other cases of chronic progressive ulceration previously labelled 'tuberculous' are in fact due to a hæmolytic streptococcus of unusual type, and would respond to treatment by zinc peroxide.

I am indebted to Sir Henry Gauvain, the Medical Superintendent of the Lord Mayor Treloar Cripples' Hospital, for his kind permission to publish the history of this case.

REFERENCES

[1] MELENEY, F. L., *Ann. of Surg.*, 1935, April.
[2] STEWART-WALLACE, A. M., *Brit. Jour. Surg.*, 1935, xxii, April.
[3] MELENEY, F. L., *Surg. Gynecol. and Obst.*, 1933, lvi, May.

AN INVESTIGATION INTO THE CONDITION OF THE BLADDER MUCOSA IN RELATION TO STONE FORMATION

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In considering the question of the aetiology of calculus of the urinary tract as the condition is seen in China, there is a marked predominance of the bladder as the commonest seat.[4, 5] To some extent this may be accounted for by the fact that, in the interior especially, X-ray and other facilities for thorough investigation are not always available. One very good reason for this is that the cost of such investigation is as much as the average countryman could earn in many months. However, even in Shanghai, where the financial stringency is not so acute, there still appears to be a marked preponderance of vesical cases when thorough investigation has been made. Our patients come to us reasonably early from the country, and it would rather appear that the condition of vesical calculus is well recognized by them and that it is appreciated that western medicine has something to offer in the way of treatment. Certainly there is no previous instrumentation, although most will no doubt have tried some native drug, often of a not unreasonable character (such as a urinary antispasmodic).

It is not proposed to discuss any further reasons for this, as it has already been done in previous papers.[7, 8, 9]. Here it is desired to draw attention to the similarity in distribution in the case of urinary tract calculus produced experimentally in rats by vitamin-A-deficiency diets. It is the experience of most workers that with such diets stones tend to form earlier and more frequently in the bladder than in the kidneys.[1, 2] It is proposed in the following pages to correlate this observation with a histological investigation of the bladder mucosa in experimental animals and in patients with calculus.

The writer has had the opportunity of carrying out such a histological examination on the epithelium of the urinary tract in rats on a vitamin-A-deficiency diet and has concluded that the changes that are found are characteristic. In the early stage there is a marked hyperplasia, and often active mitosis can be seen going on in the cells of the deeper layers. Later the epithelium undergoes metaplasia to the squamous type, and in the last stages marked degrees of keratinization are present. The changes occur first in the bladder, especially at the base and around the internal meatus. The fundus may even show no or but slight change when at the vesical orifice it is marked. The changes occur also in the ureters and kidneys, but are later in appearing, so that when present in the bladder the renal pelves may still be normal. When the deficiency diet is carried on for a long period the changes are widespread through the urinary tract and so intense as to resemble carcinoma, but in our experience it never has been possible to produce this. Fujimaki, in his experiments, claims to have produced squamous-cell carcinoma of the stomach by this means, but certainly we have never observed this or

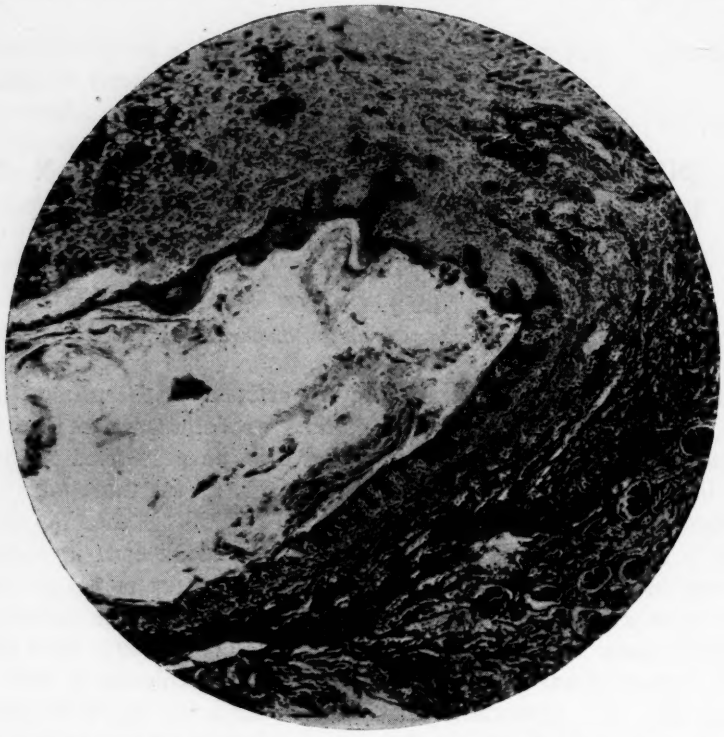

FIG. 190.—Pelvis of kidney from rat on experimental vitamin-A-deficiency diet, showing that changes are not nearly as severe as in the bladder. (× 41.)

FIG. 191.—Experimental deficiency (rat). Severe keratinization of bladder mucosa. This is the most advanced stage. (× 140.)

FIG. 192.—Experimental deficiency (rat). Bladder mucosa, showing marked hyperplasia of the epithelium and change to the squamous type. (× 41.)

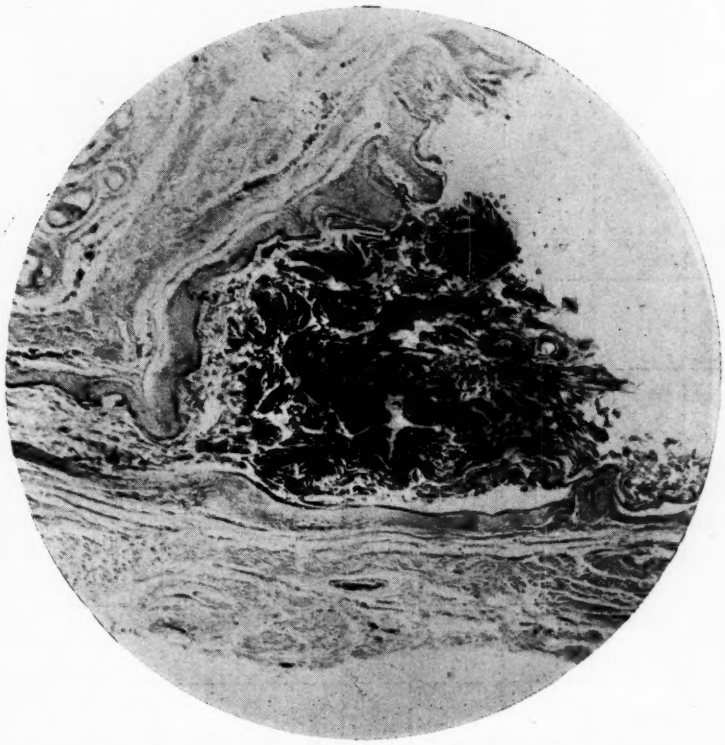

FIG. 193.—Same section stained to demonstrate the tendency for the deposition of calcium in the dead epithelium. (× 41.)

Table I.—VESICAL CALCULUS IN RELATION TO VITAMIN-A DEFICIENCY

YEAR	CASE NO.	SEX	AGE	SITE	LENGTH OF HISTORY	INFECTION OF URINE	DIET	CLINICAL EVIDENCE OF VITAMIN-A DEFICIENCY	HISTOLOGY OF EPITHELIUM	COMPLICATIONS
1934	1188	M.	10	Bladder	5 yr.	*B. coli* and staphylococcus	Green vegetables, rice, pork	Nil	Some hypertrophy of mucosa, but transitional and round-celled exudate beneath	Hydronephrosis, bilateral; syphilis
	1644	M.	14	Bladder	4 yr.	Streptococcus	Rice, vegetables, and pork occasionally	Nil	Transitional epithelium and round-celled exudate	
	1825	M.	9	Bladder	1½ yr.	*B. coli*	Not stated	Nil	Metaplasia. Epithelium squamous in type and hypertrophied	
	2171	M.	10	Bladder	4 yr.	*B. coli*	Rice, vegetables, soya bean, meat	Nil	Epithelium squamous; inflammatory reaction present	
	2331	M.	11	Bladder	5 yr.	Clear, probably sterile	Rice, vegetables, no meat	Nil	Epithelium squamous, with keratinization	
1936	3942	M.	9	Bladder	5 yr.	Clear, probably sterile	Rice, vegetables, fish, meat, pork	Nil	Epithelium squamous	Pyonephrosis
1937	720	M.	10	Bladder	7 yr.	Sterile	Vegetables, meat, eggs	Hyperkeratosis on back	Epithelium squamous, with keratinization. Many cells of germinal layer show mitosis	Hydronephrosis, bilateral
	1881	M.	11	Bladder	4 yr.	Sterile	Meat, rice, vegetables	Nil	Epithelium transitional	Hydronephrosis, right
	4790	M.	34	Bladder	6 mth.	*B. coli*	Meat, rice, vegetables	Nil	Epithelium transitional. Chronic infection	Enlarged prostate; diverticula; pyelitis
	5332	M.	27	Bladder	20 yr.	Infected	Fish, meat, eggs, vegetables	Nil	Hyperplasia of epithelium; some metaplasia at one or two points. Chronic cystitis, with early keratinization	
(Hwai	681 Yuan)	M.	23	Bladder	Not stated	Not stated	Mainly wheat	Nil	Metaplasia to squamous, and keratinization pre-	

carcinoma of the bladder, perhaps one reason being that it is difficult to keep the animals alive long enough on such a severely restricted diet. These observations are well known, [6]and a further description is unnecessary. The photographs of actual records illustrate the conditions found (*Figs.* 190–193). Simultaneous chemico-histological examinations in these cases showed deposits of calcium in relation to the desquamated and altered epithelium.

As the question of the possibility of a vitamin-A-deficiency diet having any relationship to the human condition is frequently raised, it was felt that a study of the vesical mucosa obtained by biopsy during operation might very well produce

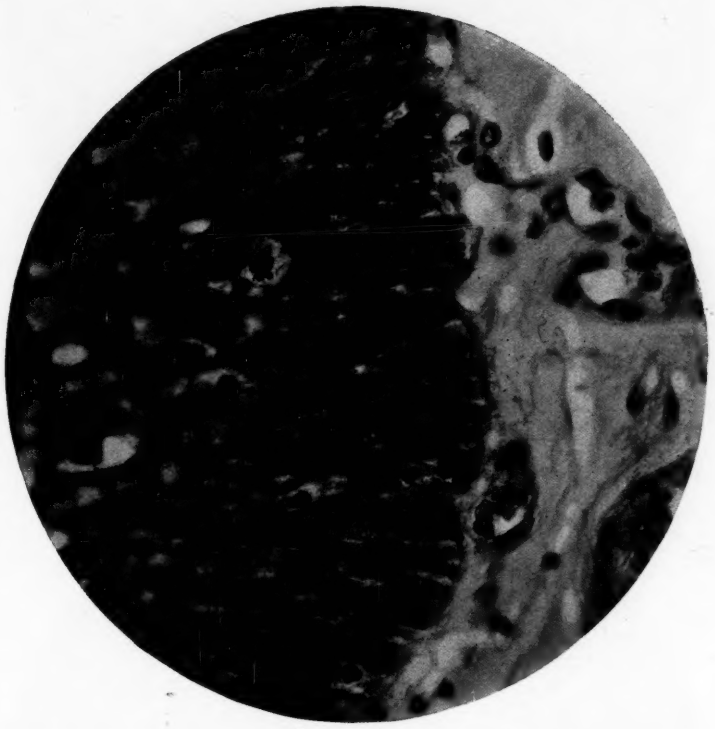

FIG. 194.—*Case* No. 720. Active mitosis. This is very similar to the biopsy appearance of the skin in clinical hypovitaminosis. (× 450.)

some evidence one way or another. It may be stated clinically that although skin conditions almost certainly due to vitamin-A deficiency are frequently seen, such patients seldom, if ever, seem to have any evidence of urinary calculus. On the other hand, patients with urinary calculus, particularly children, more often than not have a remarkably clear skin with no hyperkeratosis of any sort, nor has it been possible in most cases to discover that their diet is in any way suggestive of a vitamin-A deficiency. It was with this object in mind that biopsy was carried out where possible. The number of cases available has been small because the treatment preferred for vesical calculus has been litholapaxy. However, during the last few years some ten or more cases have been examined in this way. The findings are given in *Table I.* Biopsy specimens have also been examined in several cases where cystostomy had been performed for other reasons (e.g., prostatectomy),

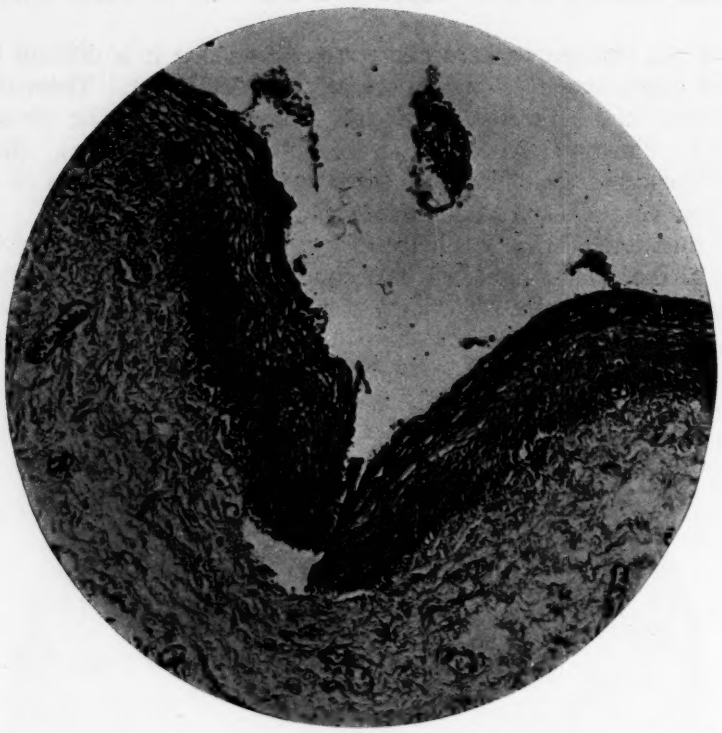

FIG. 195.—*Case* No. 2331. Keratinization. This bladder mucosa shows a metaplasia to the squamous type, with, in addition, early keratinization. (× 98.)

FIG. 196.—Normal bladder mucosa for comparison. (× 188.)

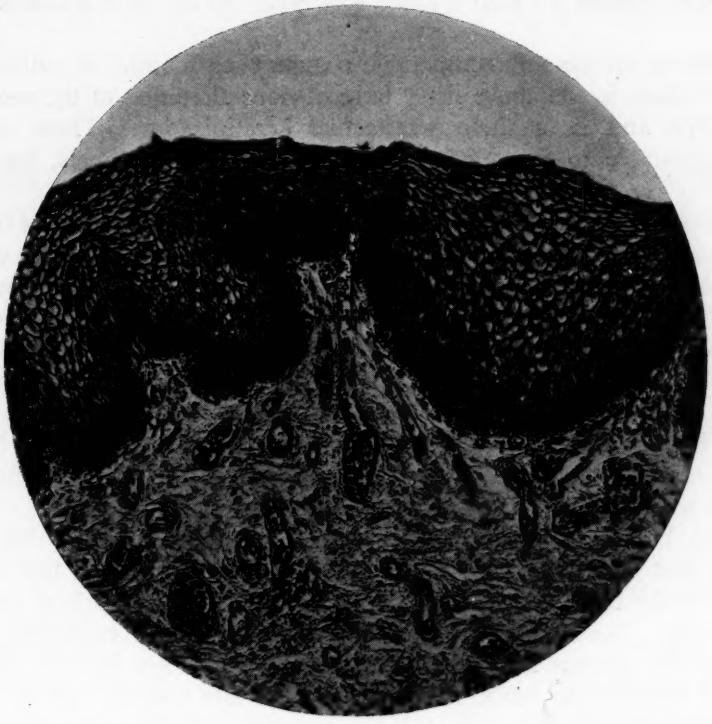

FIG. 197.—*Case* No. 720. Hyperplastic bladder mucosa changing to squamous type. For clinical details see synopsis; but note especially sterile urine. (× 100.)

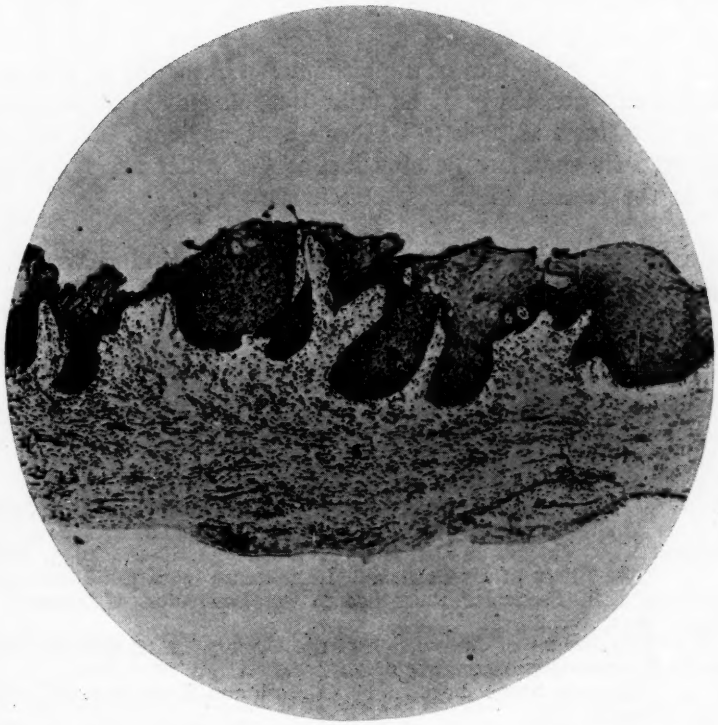

FIG. 198.—*Case* No. 1825. Epithelium shows hyperplasia and some metaplasia to the squamous type, though not as marked as in *Case* No. 720. This is regarded as an earlier stage. (× 61.)

but in no case so far has any comparable change been found. It will be seen that out of the 11 cases in the above list 7 have obvious alteration of the mucosa to the squamous type and in 4 there was actual keratinization. These changes are certainly comparable to those found in the experimental animals, but are never so intense. (*Figs.* 194–198.)

For the reasons given in discussing the experimental work, the mucosa for the biopsy was taken as near the base of the bladder as possible, but for convenience from the anterior wall. It may be of interest to mention that both on naked-eye and cystoscopic examination of the bladder it has never been possible to make out in the mucosa anything that appeared pathological. Certainly there never has been any question of leukoplakia or malakoplakia. We have even tried giving intra-vital dyes in the hope that pathological lesions in the mucosa could be 'spotted' on cystoscopy, but without success. We have not had the opportunity of studying the epithelium of the renal pelvis except in a few cases of nephrectomy for stone, and in such we found no abnormality. The question may be raised: Could not this condition be due to a severe urinary infection? In reply to this it may be pointed out that while the majority of our cases are infected, a few have sterile urine, particularly those occurring in children, as can be observed on reference to the synopsis. Further, in the experimentally produced condition, if care is taken to keep the animals in sterile cages, the urine does not become infected until quite late. Certainly the mucosal changes are already present, and in most cases the stones, too. However, even in the experimental animals stones were sometimes found before the more extreme changes had occurred, though in the final stages stones were practically always present. It was found in practice that the addition of an excess of vitamin D to the diet accelerated the formation of the stones.

It seems reasonable to suppose that in the presence of such gross alteration to the integrity of the epithelium of the bladder the formation of a urinary calculus would be facilitated, particularly if the urine were already supersaturated with the substances out of which a stone is to be formed. It is hoped that the enumeration of these findings may therefore be of value, as the writer has not seen any such investigation in the human subject described elsewhere.

I am indebted to Mr. R. V. Dent for the photographs and to Mr. W. Henderson for technical assistance with the histology.

REFERENCES

[1] FUJIMAKI, *League of Nations Report on Science of Nutrition in Japan*, 1926. Geneva.
[2] MCCARRISON, *Ind. Jour. Med. Research* xiv, 895; xv, 197; xvii, 1101; xix, 1; *Brit. Med. Jour.*, 1931, i, 1009.
[3] VAN LEERSUM, J., *Jour. of Biol. Chem.*, 1928, lxxvi, 136; lxxix, 460.
[4] THOMSON, J. O., "Urinary Stones in Canton", *Surg., Gynecol. and Obst.*, 1921, xxxii, 44.
[5] PRICE, P. B., "Bladder Stone", *China Med. Jour.*, 1934, May, 462.
[6] GROSSMANN, V., "Vitamin A Deficiency", *Wien. med. Woch.*, 1933, Nov., 264.
[7] GRAY, J., "The Effect of Experimental Interference with the Blood-supply of the Kidneys, with Particular Reference to the Formation of Stones", *Brit. Jour. Surg.*, 1935, Oct., 458.
[8] GRAY, J., "The Effects of Obstruction of the Urinary Tract, with Particular Relation to the Formation of Stones", *Ibid.*, 451.
[9] GRAY, J., "Urinary Stone Formation, Clinical and Experimental", *Chinese Med. Jour.*, 1936, l, 761.

ON THE SACCULAR ANEURYSMS OF THE INTERNAL CAROTID ARTERY IN THE CAVERNOUS SINUS

By GEOFFREY JEFFERSON

THE non-fistulous, saccular aneurysms of the internal carotid artery which develop with unexplained frequency in the intracavernous (subclinoid) portion of its course form an interesting group for study. And since chance has so ordered it that an unusual number have come under the writer's observation, an analysis of their more common manifestations can be presented, together with some suggestions concerning their clinical grouping and treatment. The pain to which these aneurysms give rise is so great that surgical aid is almost invariably sought, and by no means always then with a clear idea of the causal lesion. It behoves the surgeon to act with the discretion born of knowledge; it is hoped that the present study may in some way further that.

It is curious that whilst aneurysms are very rare in the cervical portion of the internal carotid, they are common on its intracranial portion and on the branches which form its final ramifications. Of recent years medical literature has found place for many records concerning the results, catastrophic or merely disabling, to which these pathological entities give rise. The smaller aneurysms are prone to rupture and to produce widespread subarachnoid bleeding, without giving rise to any clear-cut signs by which their exact site on the vascular tree can be recognized. The larger examples behave much more like tumours; especially do they do this after the often violent disturbances associated with their development have settled down. All aneurysms show a strong natural tendency to thrombose, and from then on to develop more slowly, or to remain stationary over long periods. The saccular (or fusiform) and non-fistulous aneurysms of the internal carotid in the cavernous sinus show these points well—an abrupt onset with signs which are ambiguous to the uninstructed (and yet so stereotyped that the next case faithfully repeats the features of the last), and then a period of stability lasting often for many years.

It will not be necessary to give a detailed account of each of the seventeen cases on which this paper is founded. Anæsthesia of some part of the face is characteristic of these lesions; they will therefore be grouped on a clinical basis in relation to the branches of the trigeminal nerve which were affected, and illustrative examples chosen. The after-histories of all the cases are known, and some of them have been followed sufficiently long for the impression that these are the most favourable of all intracranial aneurysms to strengthen into a conviction.

MATERIAL

During the past fifteen years 55 cases of intracranial aneurysm have been, at one period or another of their evolution, under the writer's care. In some the exact site of the aneurysm was never discovered, because the symptoms were purely those of subarachnoid hæmorrhage without local signature. The clinical features

of this group have been made familiar by the writing of Fearnsides and of C. P. Symonds in particular; the surgeon is rarely asked to see them, and so it will remain until he has perfected a method of treatment. The first steps in this direction have been made (Dott,[18] Tönnis,[70] and the author). In others again, the development of the aneurysm from one of the vessels which make up the circle of Willis has led to paralysis of one of the motor nerves to the eyeball; others have compressed the optic nerves or chiasma or optic tract. These last have been the subject of a special communication by the present writer, and will not be included here. Again, others lie in the cavernous sinus, and make up the group now under discussion. The cases can best be classified in the following manner:—

Apoplectic Type:

I. Aneurysms giving rise to subarachnoid hæmorrhage, but not otherwise declaring themselves 16

Paralytic Type:

II. Aneurysms with isolated paralysis of the third, fourth, or sixth nerves 10

III. Saccular (non-fistulous) aneurysms of the internal carotid in the cavernous sinus 17

IV. Basal aneurysms with affections of the visual pathways .. 12

55

It is group III which is the subject of the present inquiry.

It may be remarked that some such categorical arrangement is badly needed, not only as an exercise in clarity of clinical conception, but in order to disclose the direction in which further observation should be directed (e.g., the clinical picture of aneurysms of the basilar and vertebral arteries, and those of the more distal branches of all the vessels). The discussion of the visual defects produced by one group of aneurysms was an effort with the same end in view. The local neurological picture accompanying caroticocavernous fistulous aneurysms is another subject worthy of appraisement in the light of present-day knowledge.

PREVIOUSLY RECORDED CASES

The surgeon of a century ago was more deeply interested in aneurysms than is his great-grandchild to-day, for these were common lesions and entered much more trenchantly into diagnosis then than now. It seems to be the fact, none the less, that whereas peripheral aneurysms are becoming rarities, the intracranial type seems commoner than it was, for it is a non-syphilitic lesion which arises out of certain inborn anatomical peculiarities of the cerebral vascular tree. Its incidence, therefore, has not been affected by those improvements in anti-luetic treatment which have caused the disappearance of aneurysmal cases from surgical wards.

The first composite picture of the intracavernous aneurysm was that of Roberts Bartholow, Professor of Materia Medica in the Medical College of Ohio. In 1872 he wrote: "An aneurysm of the internal carotid will affect the sense of smell by compression of the olfactory nerve [in this he was incorrect; they rarely do so], will also cause ptosis, convergent strabismus, and a dilated pupil by pressure on the motor oculi; will produce amaurosis by direct interference with the optic; will cause congestion of the eye and swelling of the veins of the face by compression of the cavernous sinus [this is very rare, save with fistulous aneurysms], and will be accompanied by intense tic doloreux, especially in the ophthalmic division, due

to irritation of the fifth nerve. With the growth of the tumour, especially if that portion of the artery within the carotid canal be involved, there will occur noises, pulsating in character, in the ear [this is uncommon with non-fistulous aneurysms] followed by impaired hearing, and finally by complete deafness. The facial nerve may also be involved and paralysis of the face on the same side observed. Softening of the neighbouring part of the middle lobe will take place, and by reason of this, or by pressure on the crus cerebri, crossed hemiplegia will occur." It is clear that Bartholow has mixed up in this global picture the signs and symptoms of both fistulous and non-fistulous aneurysms (differentiated by Benjamin Travers in 1817), and, in any case, every single sign mentioned by him is not likely to reproduce itself in full in any given individual.

Besides papers detailing a single case, in which the old records of the Pathological Society of London are particularly rich, long papers on aneurysms of various sorts had already been brought out by Gull (1859), Gouguenheim (1866), Lebert (1866), and by Durand (1868), setting forth new cases and those collected from the literature. A better collection still was to be that of Peacock in 1875. Until that date the vertebral and basilar arteries had been thought more liable to aneurysm than the carotid, a belief which later experience has not sustained. The earliest English case was that of Sir Gilbert Blane* (1800); it will again be referred to because there were bilateral aneurysms. Only two similar cases have been reported until that of Dr. Grainger Stewart, included in this paper.

No very serious attempt to classify these aneurysms was made from Bartholow's time until well into the twentieth century, when interest was awakened by the remarkable collection of C. F. Beadles (1907), by Fearnsides' paper (1916), and by that of C. P. Symonds (1923). Harvey Cushing, in a paper consecutive to Symonds's, described some examples of intracranial aneurysm, including one of the intracavernous type. In 1926 Sosman, the radiologist to the Brigham Hospital in Boston, who had dealt with all of Harvey Cushing's material, published in company with Vogt a very thoughtful paper on these aneurysms. These authors not only described the radiological appearance of every aneurysmal case that had been admitted to the Hospital, but added sufficient clinical details to make the cases intelligible. Next, Fuller Albright (1928) recorded another example and reviewed a mixed series of aneurysms from the literature, with the partial emergence of certain clinical groupings. Recently McKinney, Acree, and Soltz (1936) reported 8 cases of saccular intracavernous aneurysm from the New York Neurological Institute, the largest single group so far brought to light, and state that the total number reported of this type is 29. The present contribution of 17 cases, much the largest individual contribution, suggests that these intracavernous aneurysms must be distinctly commoner than has been thought (cp. Viets, 1918), and that the fault has been in non-recognition.

CLINICAL TYPES

In order that the clinical signs of the sub-clinoid carotid aneurysm may be fully understood, it will be necessary to refer very briefly to the anatomical relationships

* Blane's paper was in the curiously named *Transactions of a Society for the Improvement of Medical and Chirurgical Knowledge*, 1800. It was an ironical experience for the present writer to cut the pages and open for the first time, 137 years after publication, the pages of a copy of a journal with so praiseworthy a title.

of the structures in the cavernous sinus. *Fig.* 199, made from special dissections by Professor Donal Sheehan, shows these clearly (the admirable preparations of Wolff also repay study). The curious curvature of the artery is well shown in the figure, as is the early contact which the abducens nerve makes with it. Farther along, all the oculomotor nerves (3, 4, 6) are equally vulnerable, and liable to injury by a blow-out of the vessel wall. The chief interest, as we shall see when we come to study actual case-histories, lies in the relationship to the artery of the trigeminal

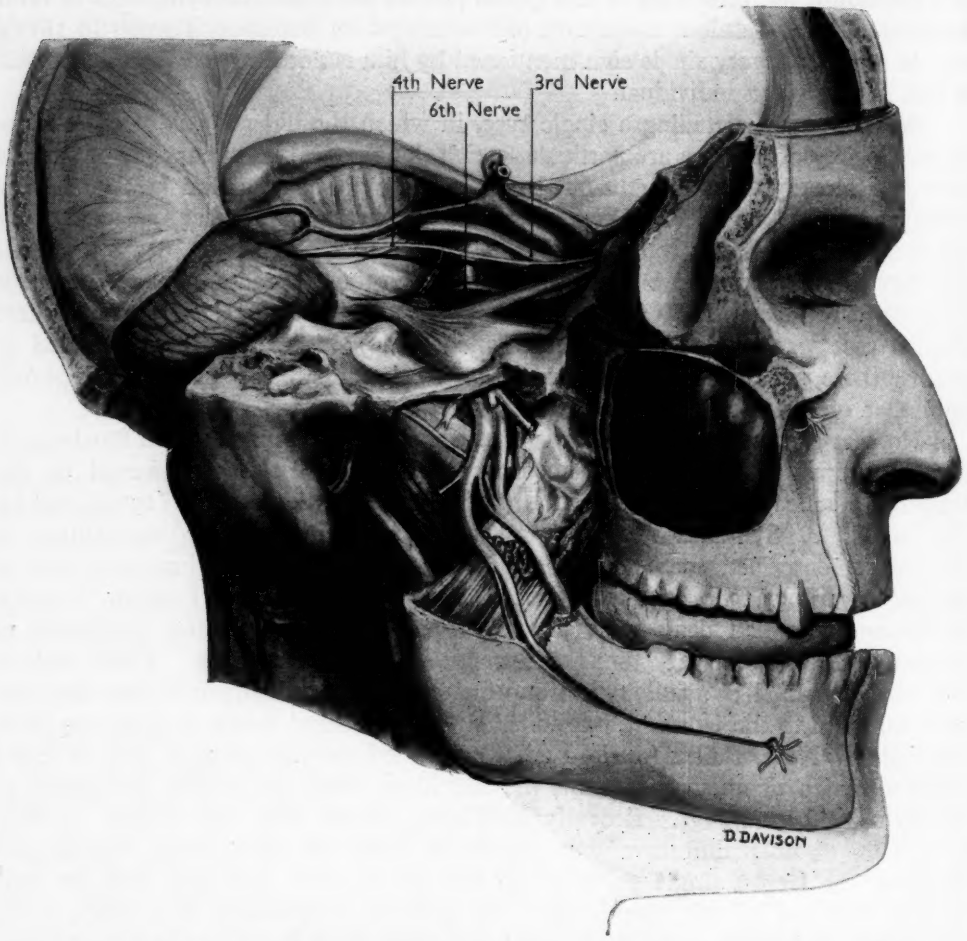

FIG. 199.—A dissection of the cavernous sinus, trigeminus, and carotid artery, specially made by Professor Donal Sheehan.

nerve, the branches of which are arranged in echelon. The third division, with the motor root, leaves the skull through the foramen ovale almost at once, so that it must escape compression, unless the artery is dilated from its point of entrance into the skull (*see Fig.* 217). The sensory root itself could also only be affected by an extremely posterior lesion, especially by a dilatation back into the foramen lacerum medium. The first and second divisions continue forward, and, given an aneurysm of the middle portion of the sinus, these two nerves will both be affected, whilst the third division escapes injury. If the lesion only involves the extreme

anterior end of the artery, at its genu, the second division escapes, leaving only the first division to suffer. Although in a wide sense there can only be one paralytic syndrome of the cavernous sinus, yet sub-groupings can be made on these anatomical grounds. That this is not a purely hypothetical conception is proved by the fact that in practice the clinical cases can be placed accurately into these groups. They are illustrated diagramatically in *Fig.* 200, and can be summarized thus :—

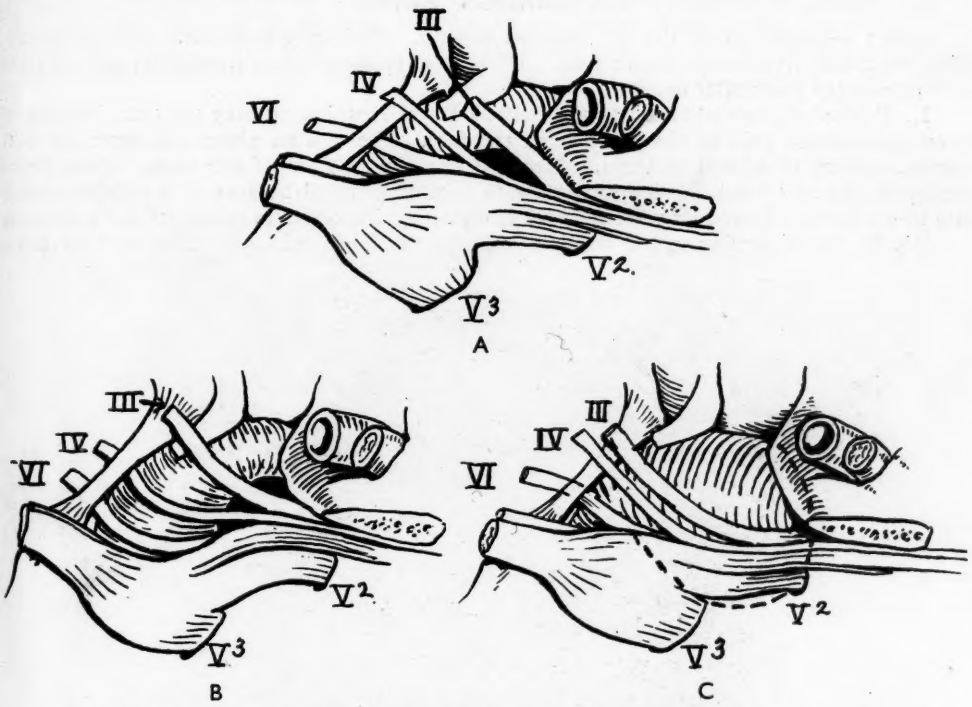

FIG. 200.—Diagrams of the anatomical syndromes of the cavernous sinus. A, Normal; B, Syndrome of posterior end of cavernous sinus (*Group A*).; C, Middle syndrome of cavernous sinus (*Group B*).

Group A. **Posterior Cavernous Syndrome.**—Whole trigeminus affected with ocular palsies, sometimes only abducens. Motor root of trigeminus affected, but may escape.

Group B. **Middle Cavernous Syndrome.**—First and second divisions of trigeminus affected, third spared. Paralysis of one nerve, usually of all nerves, supplying muscles of eye.

Group C. **Anterior Cavernous Syndrome.**—First division of trigeminus affected, the other two divisions spared. Paralysis of superior division of the oculomotor nerve, or all nerves supplying the mobility of the eyeball.

C. Foix in 1922 re-discovered the syndrome of the cavernous sinus, describing two cases of tumour there. One was unverified, the other proved at necropsy to be a carcinoma. The paralytic signs have become known, very undeservedly, as the ' syndrome of Foix '. Not only had Bartholow described it fifty years before, but in addition J. S. B. Stopford in 1917 had pointed out that theoretically lesions in this site, especially aneurysms, must produce a clearly recognizable anatomical picture.

PERSONALLY OBSERVED CASES

The 16 personally observed cases of infra-clinoid aneurysm will be grouped under the headings on p. 271; such as illustrate special points in symptomatology and treatment will be described in detail, the others will be briefly abstracted.

Group A.—SYNDROME OF THE POSTERIOR END OF THE CAVERNOUS SINUS

Case 1.—Aneurysm of the left internal carotid. Abducens paralysis. Complete left-sided trigeminal hypalgesia, severe pain. Division of sensory root of trigeminus and survival for 14 years (18 years after onset).

L. T., aged 51, was admitted to the Salford Royal Hospital on May 26, 1922, because of severe paroxysmal pain in the left side of the face. She was an obese but generally well woman, mother of several children, active and cheerful in spite of her pains. Four years previously she had been admitted urgently to another hospital because of a sudden severe pain in her head, followed by vomiting and drowsiness. No accurate record of this admission is available, but according to her own account she was unconscious for two or three days,

FIG. 201.—Photograph of *Case* 1, showing the trigeminal anæsthesia and the abducens palsy. The patient survived for 18 years.

and on returning to consciousness found that the left eye was turned inwards. This abducens paralysis persisted unaltered, but from that time onwards she began to have pain in the left side of the face. This pain was chiefly centred in the left cheek and side of the nose, in the left eye and forehead. At times the pain was paroxysmal in character, but she was rarely free from some discomfort. A year before the left nostril began to bleed at times, sometimes

severely. She consulted a rhinologist in 1921 and he performed an intranasal operation, without relief. In February, 1922, she had an alcohol injection into the left Gasserian ganglion which gave a partial cure, and on the failure of this step she was referred to the writer. (Fearnside's case also had had an alcohol injection for this misunderstood type of pain.)

On Examination.—No abnormality was found in the nose; radiographs of the skull and sinuses were negative. She had a complete left abducens palsy (*Fig.* 201). The left trigeminal area was partly and patchily anæsthetic, the corneal reflex absent on this side. It was assumed that this hypæsthesia was due to the alcohol injection, an assumption in all probability wrong. At that date the role of aneurysm as a cause of sudden and inexplicable palsy of the eye-muscles was not fully appreciated, and it fell to an exploratory operation to

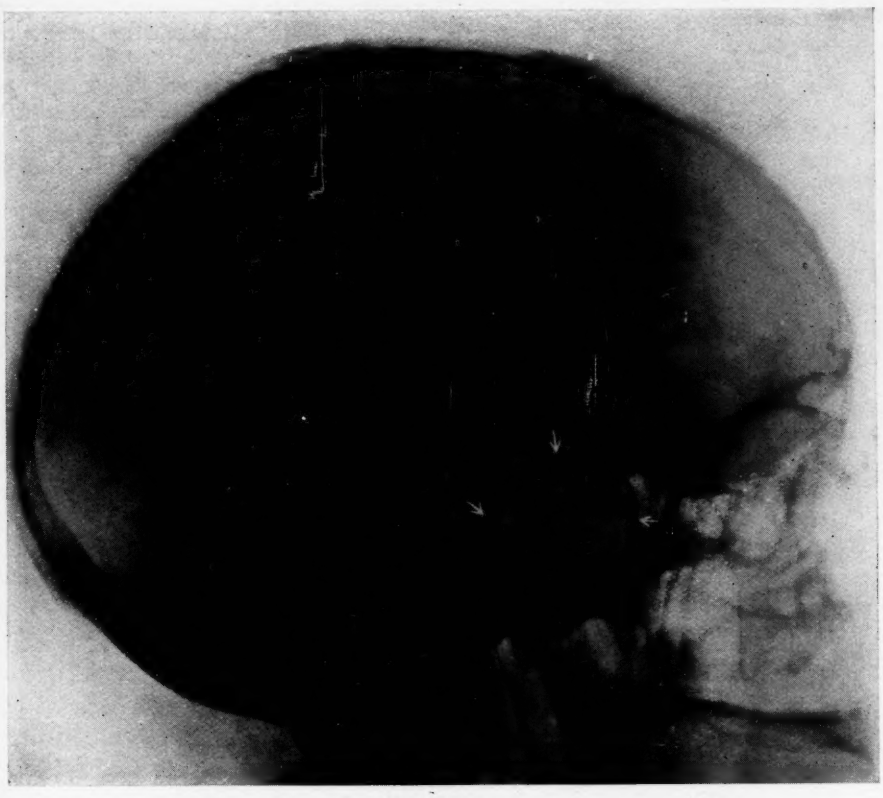

Fig. 202.—*Case* 1. Radiograph showing shell of calcification in left carotid aneurysm.

tell the full story of the lesion. The patient's remark that her face had sometimes felt numb before the injection was not given sufficient credence, for this does not happen in ordinary 'tic douloureux'. The Wasserman reaction was negative, and no other physical abnormalities were discoverable. B.P. 175/100. The visual acuity in the left eye was normal, and the nerve-heads on both sides were both entirely normal. As she was a reasonably good risk and made so much of her pain it was decided that a trigeminal root section should be done.

Operation (June 1, 1922).—Usual middle fossa approach to the trigeminal root under combined general and local anæsthesia. The dura stripped with difficulty from the bone, but the middle meningeal artery, which was large, was secured without incident. On exposing the third division of the trigeminus it became evident that an important space-occupying lesion was present, for a rounded tumour was discovered bulging outwards in the wall of the cavernous sinus, and over this the Gasserian ganglion, or as much of it as could be seen, was spread.

This tumour pulsated strongly and it was evident that we were faced with a large sacculated aneurysm of the internal carotid artery in its intracavernous portion. The trigeminal root could be seen curving back over the aneurysm and could be picked off it without difficulty. It was divided and the wound closed with a drain. Some concern had been felt during the operation that the aneurysm might rupture, but the course of this case, and of several others since, have showed that these fears were groundless.

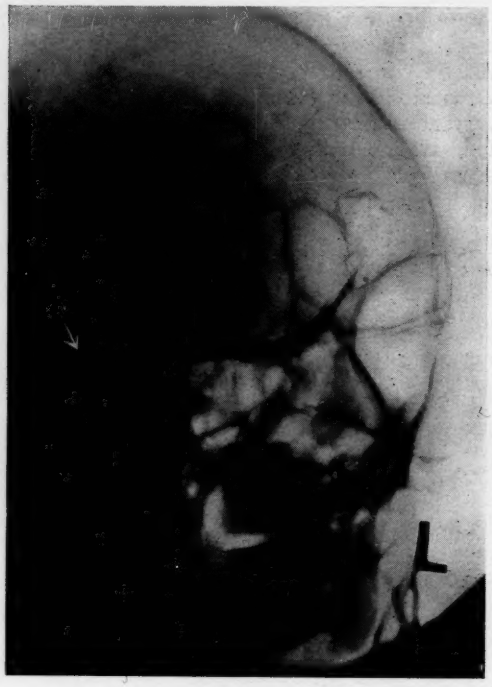

FIG. 203.—*Case* 1. Axial view showing optic canal with thinning of its lower border; calcified shell of aneurysm behind it (arrow points to aneurysm).

Apart from a neuroparalytic keratitis, convalescence was uneventful. The pains in the face were greatly relieved by division of the trigeminal root, but she was never completely cured of her pain, though never again disabled by it. Occasionally she bled ominously from the left nostril. The implication of such an epistaxis is well shown by *Case* 15.

AFTER-HISTORY.—During the next fourteen years she was kept under periodic observation, and was well able to lead an active life. In 1929 radiographs of the skull showed for the first time calcification in the wall of this aneurysm, and during the following years the shell gradually became denser (*Figs.* 202, 203). The calcified area places the aneurysm rather more anteriorly than would be expected from the observation at operation some years previously. The sac never rose high into the skull, but lay snugly on the floor of the middle fossa. Oblique views through the orbit proved that the optic foramen was not altered in size by the aneurysm, but it had eroded the lower and outward margin of this foramen.

In June, 1936, it was learned that the patient had been taken ill quite suddenly when dressed to go out and was rushed to another hospital, where she died in 24 hours. Unfortunately no post-mortem was made there.

Comment.—This case-history is, therefore, the record of a carotid aneurysm which can be accurately dated as to origin; its clinical course was observed by the writer over fourteen of the eighteen years of her illness. It is presumed that the aneurysm finally burst, though it is possible that the patient died of some other cause. Undoubtedly calcified aneurysms can rupture, because the calcification can never form a complete encasement; there must always be a weak area where the 'blow-out' joins the main trunk. The question arose at the time when the aneurysm was discovered, and again many times in subsequent years, as to whether the internal carotid artery should be ligatured. The patient was an obese, plethoric, short-necked woman, and it was decided that the risks of ligature were greater than those of non-interference. It must be remembered that an aneurysm lying in the cavernous sinus is singularly well favoured in having on one side of it at least a strong adventitious layer, the basal dura mater, which to some extent must control its tendency to enlarge, and at the same time forms a supporting sheath over it. However, that this support is not always capable of controlling the enlargement of a saccular aneurysm is well demonstrated by the next case to be described (though not the next in chronological order).

Case 2.—Aneurysm of left internal carotid. Hypæsthesia of left face, oculomotor palsy. Mental changes. Death.

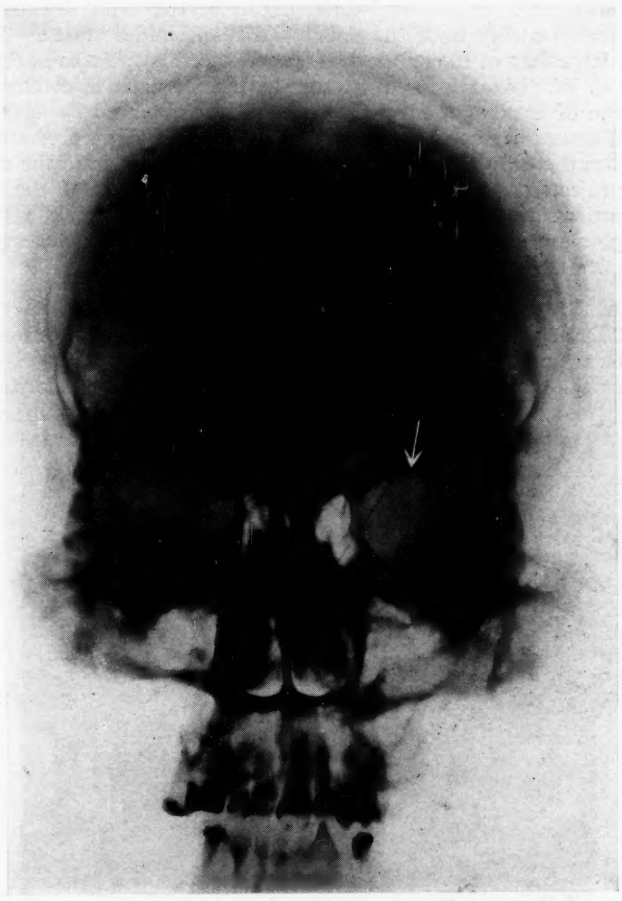

FIG. 204.—*Case* 2. Radiograph showing enlarged superior orbital fissure on the left.

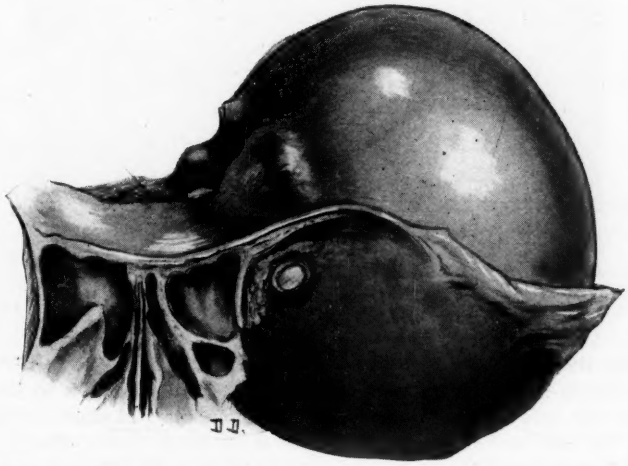

FIG. 205.—*Case* 2. The internal carotid aneurysm seen from the front. Compression of left optic nerve.

L. A., female, aged 50, admitted to the Salford Royal Hospital on November 14, 1932, with a history of numbness of the left side of the face, defective vision in the left eye, fatigue, and mental instability. This case has been described in full elsewhere, so that only the points of special importance need repetition. Radiographs of the skull showed a faint rounded shadow 6·0 cm. across arising from the floor of the left middle fossa. The most striking feature was the enlargement of the sphenoidal (superior orbital) fissure in the antero-posterior view (*Fig.* 204). E. W. Twining and the writer have since become convinced that this is a most important sign of aneurysm, and in this case it was observed by one of us for the first time. And if this fissure has been opened up by the long-standing pressure of an aneurysm, it is probable, as was the case here, that the lower and outer margin of the optic canal will be eroded. Basally projected radiographs demonstrated enlargement of the foramen lacerum medium. This patient died after eight weeks in hospital, five weeks after an exploratory operation. The post-mortem appearance is illustrated in *Fig.* 205, where the vast aneurysm is shown from the front.

Case 3.—J. A. B., female, aged 61, was admitted to the Manchester Royal Infirmary under the care of Dr. Bosdin Leech, who transferred the case to the Neuro-Surgical Service. Her health had been good apart from cholecystitis three years ago.

On November 15, 1932, whilst sweeping the floor of her house, she was suddenly seized

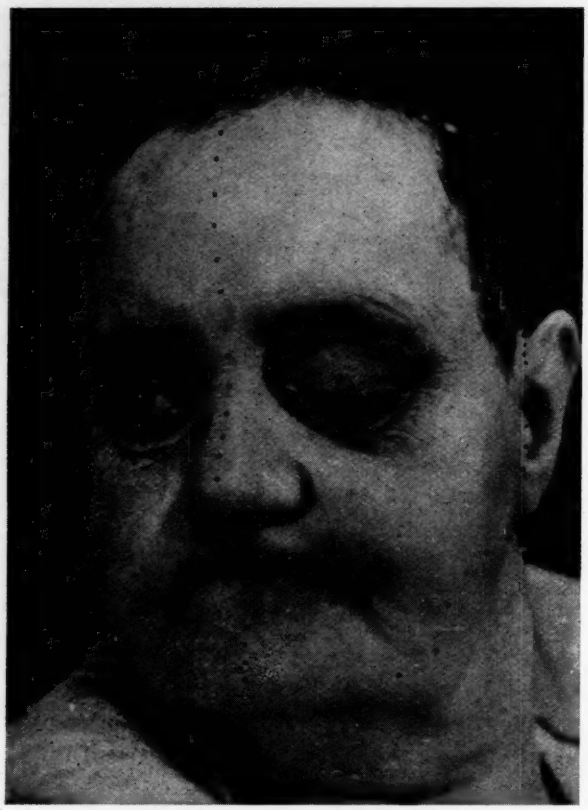

FIG. 206.—*Case* 3. Showing area of anæsthesia and ptosis.

with a terrific pain in the left temporal region, as if a knife has been thrust in. She was immediately assisted to bed, where she remained until her admission to hospital. The day after the onset the pain was worse and the left eye was found to be closed when she was roused from sleep. She had suffered from headaches for 20 years or more, always left-sided, and it is significant that 3 years previous to the present attack she had had an unexplained diplopia

that lasted for a few days. This present attack was much more severe than anything she had previously experienced, with a pulling, numb, tingling feeling in the left side of the face.

ON EXAMINATION (*Fig.* 206).—She was found to be an obese, short-necked woman with complete left-sided ophthalmoplegia and anæsthesia of the whole left trigeminal field, and weakness of the muscles of mastication on the left. Vision was not affected. No other cranial nerves were involved, no limb palsies, nor pyramidal signs. She was deaf in her left ear, and there was tinnitus of old standing. No murmur was audible on auscultation of the head. Radiographic studies showed no abnormality other than thinning of the left anterior clinoid process. Sellar contour normal. No abnormal calcification visible. The pain in the temple and face continued to be severe. B.P. 135/90. Blood Wassermann negative; cerebrospinal fluid normal.

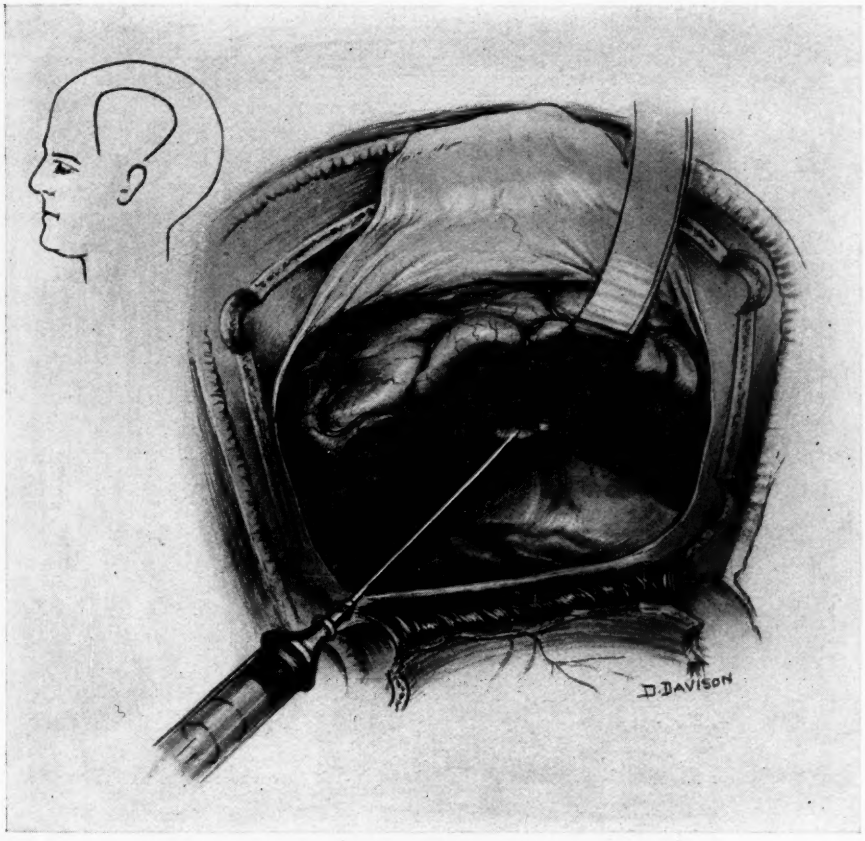

FIG. 207.—*Case* 3. Operation sketch of sac of aneurysm in left cavernous sinus.

The lesion proved to be confined to certain of the cranial nerves, with no hemispheral damage. It consisted of: (*a*) Complete left ophthalmoplegia with dilated fixed pupil, and ptosis of left upper lid, which covered the whole left globe and could not be elevated voluntarily; (*b*) Complete anæsthesia of the left side of the face in all three divisions, and motor palsy of the trigeminal root. Such a lesion could only lie at the posterior end of the cavernous sinus. In view of the sudden onset, an aneurysm was the only possibility.

OPERATION.—On November 15, 1932, in the hope of relieving her, a left sub-temporal decompression was made after cutting a small flap for purposes of exploration, the patient seated in a dental chair. The brain was rather dry. On elevating the left temporal lobe intradurally no abnormality was detected until the outer wall of the cavernous sinus was reached. This was occupied by a large rounded swelling which extended from its posterior

end forward nearly as far as the lesser wing of the sphenoid. On palpation the tumour did not pulsate, but felt doughy. A fine needle was inserted into it, but nothing could be withdrawn. A large Record needle was next inserted, and by strong suction a few flakes of old dark-brown clot were extracted. Immediate histological examination proved the material to contain no tumour cells, but ancient erythrocytes. The aneurysm was evidently thrombosed, though it might well be canalized by a small stream of blood. It was noted that there were no signs of anæmia of the cerebral areas irrigated by the middle and anterior cerebral arteries so far as could be seen through the operative approach. Usual closure. (*Fig.* 207.)

AFTER-HISTORY.—After the operation the patient was aphasic for a week, suggesting that the handling of the aneurysm had released clot to embolize. The aphasia rapidly cleared up, making œdema the more likely explanation. Her pains were definitely relieved, though

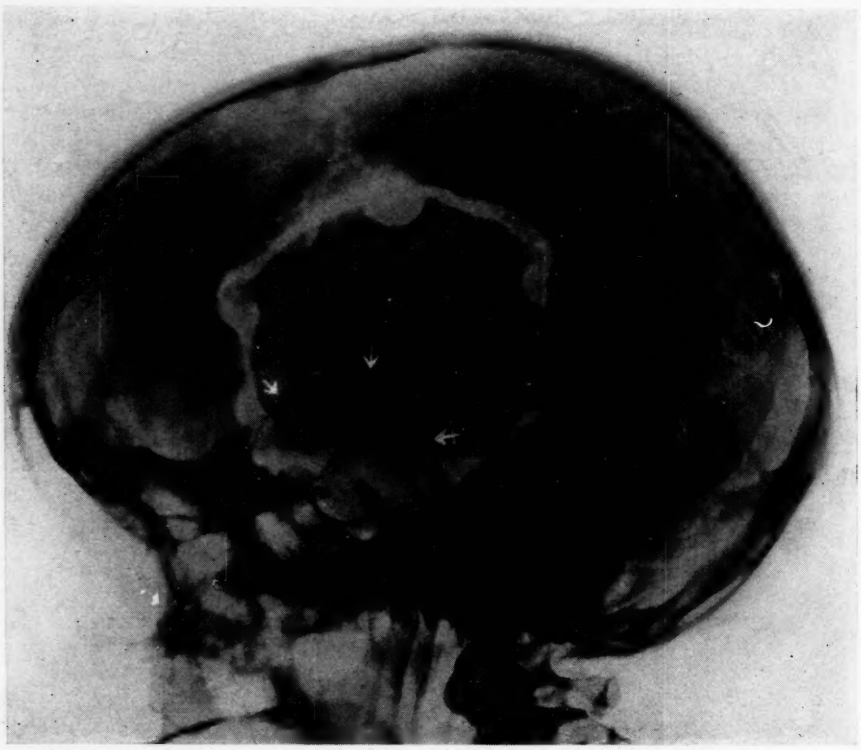

FIG. 208.—*Case* 3. Radiograph showing calcification in the sac of aneurysm five years later.

this may well have been a pure coincidence. Six months later she was making good progress, but she had had three bad attacks of pain lasting a few hours and accompanied by vomiting. During these bad attacks her forehead, teeth, and tongue became very numb. Evidently she was having occasional hypertensive attacks in the aneurysm that caused a temporary physiological block in the whole left trigeminal nerve.

This patient is alive, ambulant, with relatively little pain, in September, 1937, 5 years since the aneurysm made its appearance. She declares herself to be a very well woman, though she still has complete ophthalmoplegia, the left side of her face is anæsthetic, and the left muscles of mastication are paralysed. She has had no pain for four years. A radiograph (*Fig.* 208) shows calcification in the aneurysm sac. As she is a very obese short-necked woman ligature has never suggested itself as a practical method of treatment.

Case 4.—L. J., aged 56, referred to the Out-patient Department, Manchester Royal Infirmary, March 8, 1937. A married woman with children, she had always been healthy until two years ago, when she began to suffer from severe pain in the forehead, and her left

upper lid began to droop. A clear history was difficult to obtain owing to her mental state; her memory was bad, and she did not answer readily. She had partial paralysis of all the muscles of the left globus oculi, and drooping of the left upper lid. There was hypæsthesia of the whole left trigeminal field. This is an unverified case, but the paralysis of the muscles of the eyeball, the numbness of the face, and agonising pain leave one in little doubt but that there was an expanding lesion in the cavernous sinus. The state of this patient was even worse than that of *Case* 2, which she closely resembled. There treatment had been unavailing, and it was believed that it would be again here. Nothing, therefore, was done. In this case the outstanding feature was the psychiatric one. She was mentally deranged and her relatives dated her alteration to a time posterior to the paralysis of the eye. The not uncommon discovery of an aneurysm at post-mortem in asylum practice is referred to later.

Group B.—SYNDROME OF THE MIDDLE PORTION OF THE CAVERNOUS SINUS

Case 5.—Aneurysm of right internal carotid. Complete ophthalmoplegia. Anæsthesia of ophthalmic and maxillary division of trigeminus. Angiogram. Ligature of internal carotid artery.

E. B., aged 57, female, was admitted to the Neuro-Surgical Service of the Manchester Royal Infirmary from the Royal Eye Hospital (Dr. Duthie's case) on March 10, 1937. She had suffered from right-sided headaches practically all her life. Three years ago she began to suffer from diplopia which recovered in a day or two. Since then she had had these attacks of double vision lasting a few minutes and coming on especially when she was tired. She said that her right eye used to turn inwards. One year ago she began to have buzzing noises in her head, though no bruit could be heard clinically. Her blood-pressure was high, 220/150, and she had had one attack of nose bleeding. Three weeks prior to admission she awoke

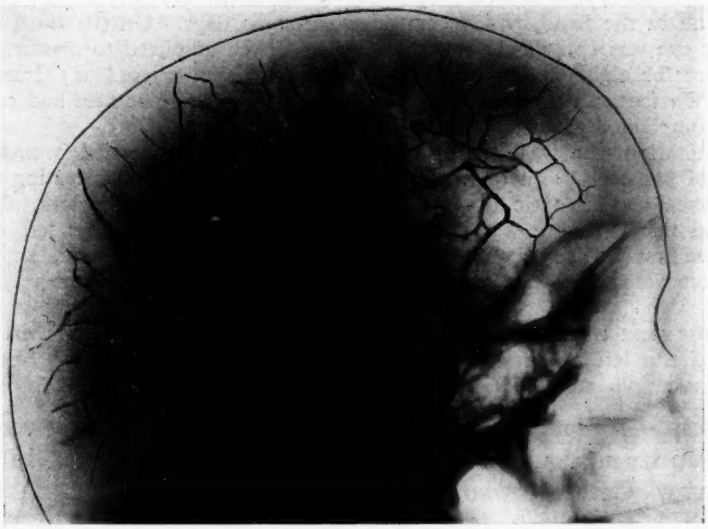

FIG. 209.—*Case* 5. Angiogram showing aneurysm in right cavernous sinus.

with a terrible headache, with severe pain in the forehead, eye, cheek, and upper jaw on the right side. This side of her face felt swollen and numb, though it looked normal. Twelve hours later the right lids were closed and remained so.

ON EXAMINATION.—She was a well-nourished woman, an intelligent and good witness. A very slight degree of exophthalmos was observed when looking down on to the lids from behind the patient. There was no œdema of the lids or conjunctiva, but total right ophthalmoplegia externa et interna was observed, with ptosis. Fundi normal, acuity normal; fields normal on repeated tests. Hypæsthesia of ophthalmic and maxillary division of trigeminus,

fading towards normal on the upper lip, was present. She was deaf in the right ear (middle ear type) and had been so for years. No pyramidal signs, slight bilateral tremor of hands, plantar responses equivocal. X-ray examination negative for sella, optic foramina, and sphenoidal fissure. Her pain was so extreme that some steps had to be taken to relieve her. Alcohol injection into the Gasserian ganglion would be unusually dangerous in a case in which the anatomical relationships were so likely to be deranged. Section of the sensory root was rendered hazardous by her hyperpiesis. It was decided to tie the internal carotid artery.

OPERATION.—On March 20, 1937, under local anæsthesia an angiogram was made, 8·0 c.c. of thorotrast being injected into the right internal carotid under local anæsthesia. A fairly good picture was obtained which demonstrated definitely an aneurysm in the right cavernous sinus (*Fig.* 209). Immediate trial catgut ligature of the internal carotid artery led to no weakness of the left arm in an hour, so a double ligature of waxed silk was added.

AFTER-HISTORY.—Six months later she was seen in the Out-patient Department and was completely relieved of her pain and very well. The right upper lid had regained power, but she still had diplopia.

Comment.—This group seems to be the rarest, and there are only 2 other examples, neither of which was verified, but the histories and clinical findings are so characteristic that we may be certain that they had aneurysms. Few cases with anæsthesia, or at least depressed sensation, in the first and second divisions are to be found in the literature (Jonathan Hutchinson, Viets).

This must mean that the aneurysms tend either to affect the whole length in a fusiform way, or to be well localized to the extreme anterior end.

Case 6.—M. E. C., female, aged 56, came to the Salford Royal Hospital on February 15, 1927, complaining of pains in the left side of the face and drooping of the left eyelid. Fourteen months previously she had been suddenly seized with very severe pain in the left side of the face and in the hand, which continued for some days. On the third day after the onset the left eye was completely closed. She had been admitted to another hospital and remained there for eight weeks, but no diagnosis had been arrived at; lumbar puncture findings and Wassermann reaction were negative. The pain in the face had continued ever since. She complained that her forehead was numb.

On examination she was seen to be an apprehensive, worried woman, constantly complaining of her head. There was ptosis of the left eye, but on elevating the lid visual acuity was normal and the optic nerve-head unchanged, as it was on the right. The left pupil was wide and fixed. There was complete left ophthalmoplegia. The left corneal reflex was absent, the first and second divisions of the trigeminus were anæsthetic, the other division normal. There were no other neurological signs; no lesion of the cranial nerves, no pyramidal signs.

During the next twelve months the ptosis improved a little and internal and external movements of the left eye returned, but not upward and downward. She refused any operative intervention, and died in 1929.

Comment.—Although the presence of an aneurysm was not verified in this case, there can be no doubt that one was present. She was seen in Manchester by Dr. Harvey Cushing, who diagnosed the presence of an internal carotid aneurysm.

Case 7.—M. J., aged 56, was admitted to the Manchester Royal Infirmary on October 6, 1928, under Dr. T. H. Oliver, on account of right-sided ptosis and pain in the head. For six months past she had been unwell, tired. Six weeks ago she began to vomit for no ascertainable reason. She also, at that date, began with diplopia. Two weeks later neuralgic pains started in the right side of the face and head, with a sensation of numbness in the same regions. At the same time the right eye closed rather suddenly and could not be opened. Her sight was not impaired in the paralysed eye. She was slightly deaf in the right ear.

ON EXAMINATION.—The patient was seen to be thin and ill. There was complete right ophthalmoplegia, internal and external, on the right side, right pupil large, fixed; the optic nerve head was normal, the visual acuity undiminished, and the visual fields normal.

The ophthalmic and maxillary divisions of the trigeminus were hypæsthetic on the right side, the mandibular division normal; motor division normal. The first division was considerably more anæsthetic than the others. There were slight but definite signs of a pyramidal lesion on the left side of the body (weakness and tendon hyper-reflectivity). A diagnosis of aneurysm was made; her bad general condition made her decide to wait for a time before considering operation.

She was discharged from hospital after five weeks. She died after sinking into coma two weeks after returning home. No necropsy.

Group C.—SYNDROME OF THE ANTERIOR PORTION OF THE CAVERNOUS SINUS

This is very distinctly the commonest group, as the 7 cases which follow testify.

Case 8.—M. G., aged 61, female, was admitted to the Salford Royal Hospital under my colleague, Dr. Don, who referred her to the writer, April 29, 1930. She had suffered all her life from periodic headaches. Her previous medical history was uneventful. Early that year she commenced with diplopia, and the right eyelid drooped a little. She attended hospital until March, when she had what she called a very bad bilious attack, with severe right frontal headache and vomiting. After a particularly severe paroxysm of terrific pain behind the right eye, the lid closed and she was not able to open it again. When she raised the lid with her finger she could see as well as usual. Her condition remained unchanged up to the date of her admission, except that the pains in her head were no longer severe.

ON EXAMINATION.—She was normal except for the alterations to be described. She was bright and co-operative. On the right side the upper lid was completely ptosed and all the eye muscles were paralysed, with a medium-sized, fixed, unreacting, pupil (ophthalmoplegia externa et interna). Vision was unimpaired. Both optic nerve-heads were normal. There

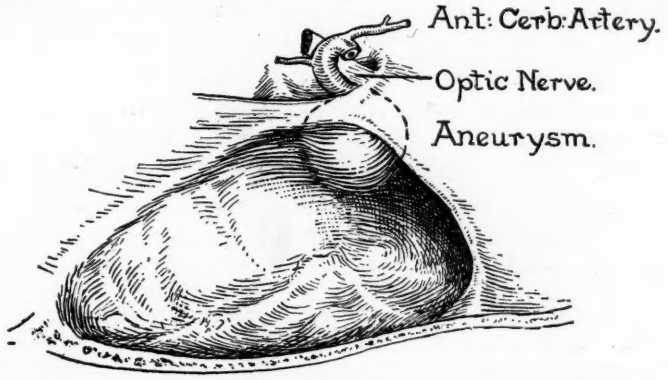

FIG. 210.—*Case* 8. Diagrammatic representation of operative findings.

was no anosmia. There was a dense anæsthesia of the ophthalmic division of the right trigeminus, the maxillary, mandibular, and motor divisions being unaffected. The remaining cranial nerves were normal. No deafness. There was no weakness of the limbs, and no pyramidal, sensory, or striatal changes in the trunk or limbs. Wassermann reaction negative. Blood-pressure 150/95. No albuminuria. C.S.F. pressure 120, no abnormality chemically or cytologically. X-ray examination negative.

In view of the sudden onset of her ophthalmoplegia it seemed certain that the lesion was a vascular one, most probably an aneurysm. It did not seem possible to link up her long history of migrainous headaches with her present attack. It was decided that the site of the aneurysm must, however, in this case be more anterior than in those previously described, because only one division of the trigeminal nerve was affected. This placed the aneurysm in the ascending part of the carotid as it sweeps upwards to pierce the dura beside the anterior clinoid process. But it could not extend far above this point, as the optic nerve was not compressed. It was possible, as an alternative, that the patient had a tumour, and an exploration was decided upon.

OPERATION.—On May 29, 1930, under combined local and general anæsthesia, a small flap was turned down in the anterior temporal region, to give access to the sphenoidal wing. It was known that the lesion must lie very close to the sphenoidal fissure. The dura was swept off the sphenoidal wing far inwards without encountering anything abnormal. Nor was the wing eroded or raised to suggest a tumour of old standing, such as a meningioma. On dissecting the dura from the middle fossa, and after plugging the foramen spinosum, the superior orbital (sphenoidal) fissure was exposed. No abnormality was detected. The dura was therefore opened, and the right temporal lobe elevated intradurally until the cavernous sinus could be seen. At its anterior end a small, rounded, freely pulsating tumour was found within the cavernous sinus, at the site of which the discovery of the aneurysm had been predicted (*Fig.* 210). Pulsation was confirmed by palpation. No local treatment was feasible. The wound was closed in the usual careful manner; the patient made an uneventful recovery, and was greatly relieved, evidently through thrombosis in the sac.

AFTER-HISTORY.—Six months later the right eye had opened, but not fully, and the abducens was now acting. The forehead was a little less numb, pinprick being recognized there.

Seen again six years later she has remained very well. The ophthalmoplegia has not completely recovered, upward and downward movements being defective, though lateral movements are good; the visual axes are sufficiently normal to allow of vision without diplopia. The palpebral fissure is still smaller on the right than on the left. Although sensation is normal in the forehead, the corneal reflex is slightly depressed. She is now 67, but very active.

Comment.—In this case it seems likely that the operative intervention was followed by thrombosis of the aneurysm, for an early improvement in her ocular palsies was manifest. Moreover, during the last six years she has remained entirely well, and free from pain. This was the fourth aneurysm in the cavernous sinus encountered by the writer, and the second operatively verified. At the present day an exploration would probably not have been made, though as things turned out it was an entirely successful method of treatment. Maybe the thrombosis which is presumed to have taken place was spontaneous and not causally related to my intervention. However that may be, the case was an important one in the writer's pilgrimage, for the exact site, shape, and size of the aneurysm were observed and confirmation obtained of the accuracy of deduction from the clinical signs which the patient presented.

Case 9.—L. C., aged 57, was admitted to the Manchester Royal Infirmary on October 14, 1933, under Dr. T. H. Oliver, who transferred the case to the writer. Married at 16, 6 children, 2 miscarriages; hysterectomy 14 years ago. Has been a hard-working, active woman, not subject to headaches. One day in February, 1933, at 6 p.m. she was coming home when she found that she had diplopia, and a headache developed. She had been quite well earlier on in the day. During the night the headache became worse, was centred in the right temple, and spread to the vertex. She commenced to vomit and her bedroom had to be darkened. She had a great deal of neuralgic pain in the right cheek back to the tragus. According to her own account, both eyes closed during the next few days, but the left opened within a week. The right remained closed, and the lid had to be elevated with the finger if she wished to see out of it. Both eyes were turned inwards originally. The right upper lid remained ptosed for three months before it started to elevate a little. She was in bed at home for three months. Her limbs were unaffected.

ON EXAMINATION.—She was a well-looking woman on admission, with incomplete ptosis of the right eye. She had had severe pain in the right temple since the onset of the illness. The right abducens was completely paralysed. The internal rectus acted fairly well but not perfectly, inferior rectus perfect, superior rectus partly paralysed, the globus oculi turns up a little but not fully. The left eye movements were normal in range, and it was difficult to know how much credence to attach to the statement that the left external rectus, and perhaps the left lid, had been paralysed as well as the right. There was no sign of it later. Both optic

nerve-heads were normal, acuity normal, visual fields normal. There was no proptosis, no pulsation in the globe. No bruit on auscultation. No anosmia. The trigeminal areas were normal, save the right corneal reflex, which was insensitive to the camel-hair brush or pin head. She would admit to no hypæsthesia in the remaining territory of the right ophthalmic division, but her forehead had been numb on this side a few weeks ago. The second division, where she had shooting pains, was certainly not anæsthetic. Other cranial nerves were normal. There were no pyramidal or sensory changes in the trunk or limbs. X-ray examination negative. Wassermann and cerebrospinal fluid findings negative. Here again was a female patient with a dramatically sudden onset of local, unilateral pain in the head, followed within a few hours by an oculomotor and abducens palsy. It is probable that she had originally a complete ophthalmoplegia, but when first examined the third nerve, at least, was recovering. None the less, there was evidence of paralysis of more than one of the nerves controlling ocular

FIG. 211.—*Case* 9. Angiogram of distal aneurysm of internal carotid.

movement on the right side. Oculomotor paralysis commencing in this manner is almost invariably aneurysmal in origin, and when more than one nerve is affected the aneurysm is usually in the cavernous sinus. In favour of this situation was the absence of her right corneal reflex, the only sign still present of any trigeminal involvement.

OPERATION.—It was decided that a thorotrast injection should be done. On Nov. 11, 1933, under local anæsthesia, an angiogram was made by injection into the right internal carotid. An excellent picture was obtained after injecting 14 cm. of thorotrast (*Fig.* 211). This shows a large aneurysm 2·0–2·5 cm. in diameter at the level where the internal carotid pierces the dura to enter the cranial chamber. It is remarkable that the optic nerve should have escaped. Judged by the size of the thorotrast shadow it was unlikely that the aneurysm contained much clot. It was resolved that the internal carotid artery should be tied. This was done at once with three fine strands of waxed silk which did not entirely occlude the lumen. Dynamometer readings were taken hourly after ligature ; the left hand grip remained perfect.

AFTER-HISTORY.—Convalescence was smooth but for slight infection in the wound in the neck, possibly due to spilling thorotrast into the tissues. The patient has been kept

under observation since the operation, and was very well in July, 1937, 3½ years later. Her oculomotor palsies have never completely recovered, especially abducens. She rarely has any headache, and can do a full day's housework.

Case 10.—E. S., female, aged 48, was seen at the Blackburn Royal Infirmary with Dr. Fairweather on July 14, 1933. The patient was generally healthy and her previous medical history was without relevant incident, apart from severe periodic frontal headaches of many years' standing. Nine days ago she was suddenly seized, without warning, by intense, almost unbearable pain in the right side of the head which induced vomiting. She went at once to bed and remained in agony for three days, unable to eat, continually troubled by vomiting, whilst the pain continued. The next day after the onset the right eyelid closed. After a week at home she was admitted to the Blackburn Royal Infirmary.

When seen there she had complete right-sided ophthalmoplegia with ptosis of the right eye; the right pupil was wide and fixed. Both optic nerve-heads were pale, no choke, acuity normal, visual fields normal to confrontation. She complained of numbness of the right forehead, and the ophthalmic division of the trigeminus was found to be completely anæsthetic, and the corneal reflex was absent. The remaining cranial nerves were normal. No palsies of limbs. Wassermann reaction negative, blood and cerebrospinal fluid. B.P. 170/100. Radiograph of the skull showed a rather large pituitary fossa and the posterior clinoids were ill-formed. No aneurysmal shadow was visible.

The patient made a good recovery with rest in bed, and three years later was able to do her housework, though the oculomotor paralysis had not completely recovered.

Case 11.—E. J., aged 76, was referred to the writer by Dr. Pringle on February 13, 1933. Until two years previously her health had been excellent. She was married at 23 and had five healthy children.

Two years ago she began with pain over the right eye and ptosis; the onset was sudden when out walking, it felt to her as if a blood-vessel had burst. Complete right-sided ophthalmoplegia, interna et externa. Cataract in right eye, no vision. Depression of right corneal reflex was all that remained of what must once have been an anæsthesia of the ophthalmic division at least. B.P. 255/140. Slight albuminuria. W.R. negative. Radiographs of skull and optic foramen normal. No treatment was undertaken because of her age and hypertension. She belongs to the senile, arteriosclerotic group, which is a small one.

This patient is alive and well, 6 years after the development of her aneurysm. Dr. Pringle reports that her headaches have disappeared and she goes about as usual. Her right eye is still closed by ptosis (September, 1937).

Case 12.—E. P., female, aged 22, admitted to the Neuro-Surgical Service, Manchester Royal Infirmary, June 26, 1936, referred by Mr. Rayner, of Preston.

A well-developed, healthy girl who had had no serious illnesses previously. Two years ago she awoke suddenly one morning with severe throbbing pain over the left forehead as if her head were being split open; slight movement caused the pain to increase. Gradual improvement, but recurrence of pain at menstruation since that time. Ten weeks ago sudden severe pain over the left eye, passing up on to the vertex, returned whilst sitting in a cinema; she vomited copiously. The left upper eyelid began to droop the same evening. Two days later she was admitted to Preston Infirmary under Mr. Rayner. Neck stiff; yellow fluid on lumbar puncture. She had complete ophthalmoplegia interna et externa, with numbness of the left forehead and absent corneal reflex on that side. When examined by the writer, the upper lid had recovered tone and was half open after two months; ocular movement was returning. Optic nerve-heads normal, vision 6/6. Visual fields normal. X-ray examination—porosis of left anterior clinoid. W.R. negative. A diagnosis of aneurysm was easily arrived at.

On July 14, 1936, the left common carotid was exposed and radiographs taken after 16 c.c. and 20 c.c. of thorotrast had been injected. Good pictures were not obtained, but overlying the sella was a localized shadow of thorotrast that corresponded exactly to the supposed site of the aneurysm. It must have been largely filled with clot.

Owing to the patient's spontaneous recovery and her complete satisfaction with her present state, the carotid artery was not tied. This may have to be done later, but in the meantime the patient is well except for occasional pain in the left fronto-temporal region. She is able to work.

Case 13.—D. D., male, aged 53. Attended the Neuro-Surgical Service of the Manchester Royal Infirmary, February 8, 1937, complaining that he could not open the left eye and of pain in left eye, forehead, and nostril, with a numb feeling on the forehead. Three months ago during influenza a severe headache developed, followed during the night by complete closure of left eye. After five weeks the eye began to open again. When seen he had complete ptosis, abducens palsy, and partial paralysis of the oculomotor and trochlear nerves. No anosmia, vision normal. No pyramidal signs. This patient had at the time of examination no demonstrable anæsthesia of the trigeminus, but the distribution of the original pain and numbness pointed very clearly to affection of the ophthalmic division. No treatment was undertaken, as he declared himself to be making a good recovery.

Case 14.—B. R., female, aged 37, married, with healthy children, was referred by my colleague, Dr. Fergus Ferguson, with paralysis of the right eyeball. He has kindly permitted me to see and record this case. The patient had an operation for prolapsus uteri early in 1937, and on recovering from the anæsthetic had a severe pain in the right occiput and in the right eye. This lasted for ten days, when she heard something snap suddenly in her head, the whole right side of the face immediately became numb, and the right upper lid closed. She declares that all of this happened within a minute or so, and that her pain then left her and has not returned. She had no neuralgic pains or pain referred to the face at all; it was just numb. Next day the anæsthesia had receded from the face, except the forehead.

On examination two months later there was complete internal and external ophthalmoplegia; pupil mid-dilated and fixed; anæsthesia of the ophthalmic division of the trigeminus, including the cornea, pallor of the right optic disc; acuity, R. 6/18, L. 6/6. No field defect could be made out. Radiographs of the right optic foramen showed thinning of the lower and outer wall of the foramen and a crescentic defect in the medial margin of the sphenoidal fissure, characteristic of aneurysm (*Fig* 212). In the lateral view the right clinoid process was elevated and there were some scattered patches of parasellar calcification, no doubt in the wall of the aneurysmal sac, though the outline of the latter could not be discerned (*Fig* 213).

Comment.—Judged on the criterion of the bone changes, it is likely that this aneurysm must have been present for some time before it gave its first signal, and a peculiarity of this case is that the pain ceased when ocular paralysis occurred. As a rule pain becomes severe at that moment: the reason for the reversal of this custom is a matter for conjecture. This patient has been re-examined on two occasions since: her condition is good, she has no pain, and everything points to the aneurysm being stabilized. Possibly her internal carotid artery ought to be tied, but she is so well that one finds it difficult to insist on this step.

The next case is of a different order, for it concerns a young man who had been exsanguinated by severe hæmorrhages from the nose. In this patient none of the neighbourhood signs of aneurysm were present, yet the case must be included because it illustrates a possible variant. It recalls the cases of Guibert and of Birley and Trotter.

Case 15.—T. H., male, aged 34, was operated upon on September 21, 1937, by Mr. Neville Young for chronic ethmoidal and sphenoidal sinusitis, with polypi. No special feature was disclosed at the intervention, which included the opening up of the sphenoidal air sinus. He went home from the nursing home, but on October 8, 1937, he had a violent hæmorrhage from the nose, a pint of blood being lost in two or three minutes. This was repeated on four more occasions during the next week, and his condition became so grave that two blood transfusions were given. Finally after a particularly severe outburst of bleeding, which pushed a pack out of his nose, we decided to tie the internal carotid artery. It was assumed after careful deliberation that the left internal carotid artery must be leaking into the sphenoidal air sinus, and of course, if it was, an aneurysm must be present. Digital compression for ten minutes caused no aphasia or weakness of the hand grip. The left internal

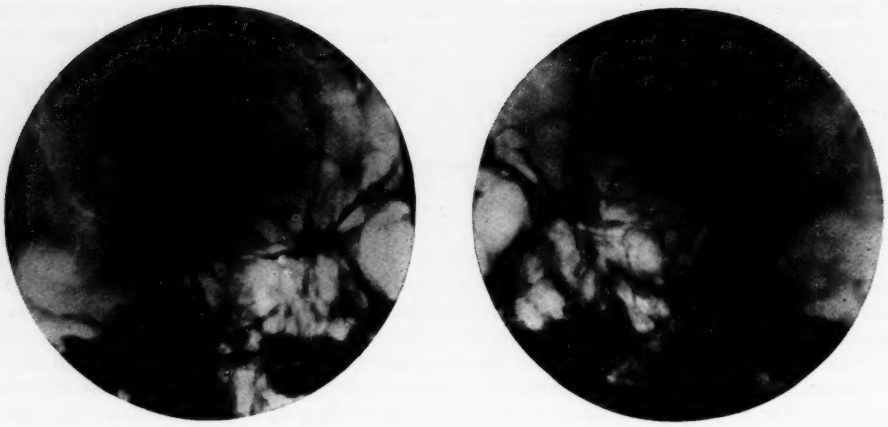

FIG. 212.—*Case* 14. Optic foramina showing thinning of lower margin of optic foramen on affected side (right) and slightly enlarged sphenoidal fissure.

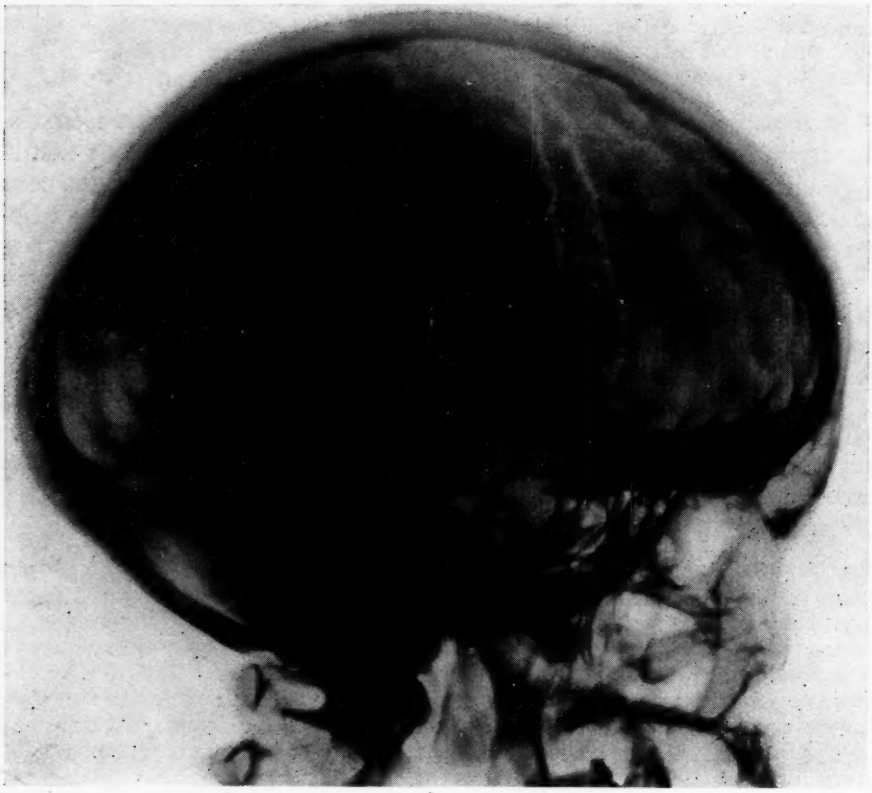

FIG. 213.—*Case* 14. Radiograph showing parasellar calcification in carotid aneurysm. Note the elevated anterior clinoid process.

carotid artery was temporarily tied under local anæsthesia; after waiting for half an hour a silk ligature was applied. That night and next day the patient was very well, but 36 hours after ligature, when he awoke on the second morning, he could not speak nor move his right arm. There was slight facial weakness. The leg was not greatly affected, he could lift it off the bed and the plantar response was flexor. He was quite alert mentally. As he was so extremely anæmic, in spite of his transfusions, a third blood replacement was given in the hope of improving his circulation generally. The next day he was the same, but on the fourth day after ligature he began to move the arm again, and by the eighth he had regained finger movement, and was beginning to talk. At the end of two weeks he was still slightly aphasic, but could read and write, though not as perfectly as formerly. When the palsy developed in this case the question of removing the ligature arose, but the temptation, if such it was, was resisted. There was no further bleeding, and he left the home again three weeks after carotid ligature with slight speech difficulty only. It transpired that there was a luetic tinge to this case: many years before he had had a positive Wassermann reaction, though not now.

Comment.—This case illustrates very well the anxieties and possible penalties that attach to carotid ligation on the left side. Aphasia and paresis do not run equally and parallel, for the former may be greatly in advance of the latter. This patient, for instance, for two or three days could not speak at all, and yet movement of the right limbs and face was not far below normal. In the end, happily, the speech deficiencies cleared up, but he talks more slowly than he did, and is sometimes at a loss for a word; especially is spelling bad when writing. In this case the aneurysm must have broken through medially into the sphenoidal air sinus, its passage having been facilitated by the ordinary manipulations of exenteration of the sinuses.

BILATERAL INTRACAVERNOUS ANEURYSM

Sir Gilbert Blane's case of bilateral aneurysm has already been mentioned. It has been included by Sattler in his great collection of arteriovenous aneurysms. There is nothing in the description of the case or of the dissection to suggest that this was a fistulous aneurysm. Blane's patient was a lady 69 years old; five years previously she was "suddenly seized with a fit of giddiness and dimness of vision, succeeded by acute pain in the forehead, which remained for some time. The indistinctness of vision continued for six months. . . . She had a similar attack two years after the first, from which also she recovered to a certain degree." Finally she became insane, "and this attack seemed to have been brought on, or at least aggravated, by strong emotions of joy and vanity upon her daughter marrying a man of very high rank." She died on June 30, 1792, in a state of "feverish mania". A dissection was made and is thus described: "But the morbid appearance in this case which was so singular, and to which the symptoms of complaint seem chiefly referrible, was two bulbs about five-eighths of an inch in diameter, filling up the hollow on each side of the sella turcica, which were evidently dilatations of the carotid arteries, and from these being filled with laminæ of coagulated blood, there could be no doubt of their being aneurisms of those arteries. The dissection was made by Mr. Hunter, assisted by Mr. Home, in the presence of Dr. Jenner and myself, and all concurred in opinion, that these tumours were aneurisms."

I have not been able to find an account of any other bilateral aneurysms save two, neither fully described. The first is that of Sosman and Vogt. This concerned a woman 62 years of age with pain in the left side of the face, left facial palsy, bilateral deafness, and diplopia. Her blood-pressure was 225/100. There were

arteriosclerotic changes in the retina. X-ray examination showed calcification in both internal carotid arteries, and at their first forward bend there were bilateral bulges so that the arteries rose above the level of the centre of the pituitary fossa. The findings were compatible with small bilateral aneurysms of the internal carotid arteries.

The second forms an illustration (Fig. 28) in W. E. Dandy's paper on carotico-cavernous fistulæ. There was a huge aneurysm on one side, a smaller one on the other; between them they had destroyed the floor of the skull. I owe *Case* 16 to the courtesy of Dr. Grainger Stewart, having happened to witness the post-mortem examination by Dr. Greenfield.

Case 16.—Bilateral carotid aneurysm, clinical signs on left side only. Necropsy.

E. L., female, married, aged 72, was admitted to the National Hospital on February 27, 1936, complaining of severe pain and numbness of the left side of the head and in the left eye, together with drooping of the left upper lid. She had had 13 children and was always a healthy and vigorous woman. A year ago she had a bad fall, bruising the left side of the body, face, and front of the chin. She was ill for two months afterwards. A month ago she woke up with a bad left-sided headache which lasted all day. In the evening she had

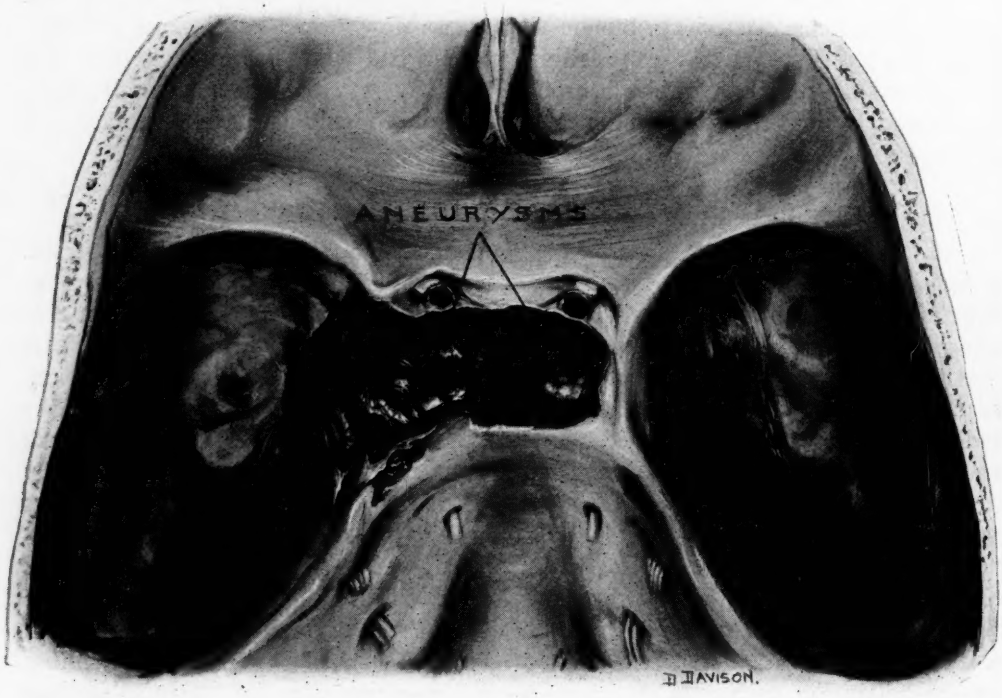

FIG. 214.—*Case* 16. Bilateral aneurysms of the internal carotid artery.

a violent attack of vomiting, and the trigeminal pain, which was to persist till her death, commenced. The pain involved the left forehead, left upper lid, globus oculi, and temple—a creeping, tugging pain, "sometimes burning like fire". It was continuous. From that day onwards the left lid drooped, and she always kept that eye closed. Examination showed the presence of an incomplete ophthalmoplegia, affecting the left 3rd, 4th, and 6th cranial nerves. The right pupil was slightly larger than the left; both were small and slightly irregular. Fundi normal. Visual acuity 2/24 right and left. No anosmia. Corneal reflex

almost absent on the left. Hypalgesia and loss of recognition of light touch over the whole left trigeminal distribution, more marked in the first and second divisions. Weakness of the left masticatory muscles. Right trigeminus normal. There were no other neurological signs. Blood-pressure 160/120. There was no exophthalmos. X-ray examination showed enlargement of the sella turcica and erosion of the dorsum sellæ. Her condition rapidly deteriorated, and after an attack of coronary occlusion she died on March 20, 1936.

Comment.—The diagnosis was aneurysm of the internal carotid artery in the cavernous sinus. The necropsy, witnessed by the writer, proved this to be correct. Dr. Greenfield discovered bilateral aneurysm (fusiform type) of the internal carotids, that on the left considerably the larger (*Fig.* 214), and indeed that on the right was a quite unexpected discovery, for it had given no sign. The vessels were arteriosclerotic, and much more abnormal than in the majority of the unilateral cases just described. Although she had had a fall, there was nothing to indicate that she had an injury to the skull, and no fracture was found. She was reasonably well after the accident for ten months, before the sudden outbreak of the symptoms that heralded the formation of the aneurysm. These were of fusiform rather than saccular type, bulging the dura on the sides of the sella outwards.

The reader may recall that in *Case* 9 the patient insisted that her symptoms had originally been bilateral, and it is conceivable that she had two aneurysms, the left one having ceased to give any signs.

CLINICAL EVOLUTION

The cases recorded above illustrate the course of events which characterize these lesions. Of the 16 cases in this series 5 have died, two quickly (*Cases* 7 and 16), one $3\frac{1}{2}$ years (*Case* 6), one 5 years (*Case* 2), one 18 years (*Case* 1), after the development of the aneurysm. Of the remainder all are able to do their work except the one patient who is mentally afflicted (*Case* 4), and one (*Case* 11) who is enfeebled by age.

In the main the onset has been sudden, but it is not invariably that the full picture presents itself immediately (e.g., *Case* 2). In general, there has occurred without any previous warning, or with prodromal discomfort that may cover some weeks, an excruciating neuralgic, sharp pain over one side of the head, over the eye, and over varying extents of the face. The pain is so sudden, so violent, that he (or more usually, she) thinks that a violent blow has been struck, and many patients have said that they were sure that something had given way in the head. Within a few hours some form of oculomotor paralysis develops, usually of the third nerve, sometimes of all the nerves supplying the muscles of the globus oculi, because the hernia rapidly comes into contact with the sinus wall. Although this apposition has unfortunate immediate results for the patient, the secondary dural envelope thus obtained is an important factor of safety. Compression of the wall of the cavernous sinus by the aneurysm leads inevitably to something else—compression of the gasserian ganglion, or more properly of one or more of its branches (*see Figs.* 199, 200), according to the situation of the lesion in the anterior, middle, or posterior parts, or in the whole length of the sinus. The aneurysm is often retained by its secondary dural envelope, and does not increase much in size. More rarely it may strip the dura from the base of the skull and spread into the floor of the middle fossa, so as to form a mass occupying the whole of it (*see Case* 2).

The grouping of the cases on an anatomical basis, depending on the territories of supply of the branches of the trigeminal nerve, needs no further emphasis here. It is this anæsthetic accompaniment of an ocular muscle palsy which makes the diagnosis of an infraclinoid aneurysm a matter of the utmost nicety and precision. Referring back, for a moment, to Bartholow's description, we may observe that in none of the present series was anosmia present (save in *Case* 15, where an ethmoidal operation had really been carried out), that all but one (*Case* 15) had ptosis, pupillary alterations, and various ocular palsies. In only one (*Case* 2) was vision interfered with except by loss of accommodation. In none was there congestion of the eye or swelling of the veins of the face, though the least trace of non-pulsating exophthalmos was occasionally present; it is generally so slight that an ophthalmometer is needed to measure it. All had intense pain, not like tic douloureux, for it was continuous and not spasmodic, though referred, more or less accurately, to the distribution of one or more of the branches of the fifth nerve. Only rarely was there tinnitus, and those who were deaf had some recognizable alteration in the middle ear. This may well have been due to compression of the Eustachian tube by a fusiform dilatation of the artery far back into the foramen lacerum medium. The facial nerve was not involved in any case; when present it might be due to deep erosion of the petrous bone, or, more probably, to tension on the great superficial petrosal nerve through the dural stripping caused by the aneurysm, and consequent damage to the seventh nerve at its genu where the petrosal joins it. Lastly, " softening of the middle lobe of the brain " : none of the present series had a hemiplegia, though one or two (*Case* 2, etc.) showed slight pyramidal signs. The mental changes, undoubtedly due to defective cerebral vascular irrigation, have already been referred to. Thus corrected, Bartholow's description may be allowed to go into currency once more, but it would be more economical to suppress it, and to sketch the picture under the terser classification already suggested.

The Fate of the Cranial Nerve Palsies.—There is in general a tendency towards recovery after the original ictus which produces the paralysis of the ocular musculature and interrupts conduction in the trigeminal nerve. As for the latter, after a few months little may remain of an original anæsthesia save an absent corneal reflex. Except as regards pain, the sensory component of the cavernous syndrome is unimportant to the patient.

The oculomotor part of the lesion is not only more obtrusive to the observer's eye, but more disturbing to the patient. There is no need to discuss the diplopias that result from paralysis of the various muscles of the eyeball, the more so since the third-nerve injury is complete enough as a rule to produce a ptosis which shuts out vision on the affected side. It is during the process of recovery that the most interesting observations are to be made. In none of the writer's series have all the muscles returned to normal. The common event is for the closed upper lid to open by degrees as the aneurysm shrinks or as the nerve recovers from its first violent stretching. The globus oculi regains lateral movement, inwards and outwards, but not upwards and downwards to the same extent. This paradoxical result—recovery in the levator palpebræ and rectus internus and externus—has been observed in experimental lesions of the third nerve in monkeys (Mahoney and Sheehan, 1936). The re-opening of the eye, never sufficient to restore the full width of the palpebral fissure, but enough to restore vision, must be due in

part to the activities of the sympathetic on Müller's muscle. The experiments of Mahoney and Sheehan have demonstrated the decreasing importance of this latter in primates as compared with cats, who are able to elevate the lid within twenty-four hours of section of the oculomotor nerve. The fact that recovery in the tone of the upper lid in man is accompanied by a return of function in the recti proves that it is due chiefly to regeneration rather than to the action of sympathetically innervated smooth muscle. The pupil in these cases, whilst not reacting to light, has shown great variations in size. The classically widely dilated pupil has not always been seen. One must assume damage to the sympathetic dilator fibres to the pupil which run from the carotid plexus to the ophthalmic division of the trigeminal nerve, as well as to the parasympathetic constrictor fibres in the third nerve. The sympathetic supply might be damaged either by pressure on the wall of the dilated carotid, or by the thrust of the aneurysm against the ophthalmic division of the fifth nerve (most likely the latter explanation is the correct one).

The reason why the lateral movements recover better than the vertical requires a few words of inquiry. The best explanation is that it is due to the crossing of axons during regeneration. If this takes place the eye will move out by the action of the abducens, where no misdirection of nerve-fibres is possible, and inwards by means of whatever proper fibres have reached the internal rectus. Misdirection of fibres in the superior and inferior recti will, on the other hand, cancel out one another's action. This application of Stopford's work on peripheral-nerve regeneration has been suggested by Ford and Woodhall, and seems to the writer the most probable explanation of a puzzling phenomenon.

DIFFERENTIAL DIAGNOSIS

Whilst the supraclinoid aneurysms most commonly cause a paralysis of one cranial nerve (usually the third, more rarely the sixth), occasionally more than one is affected. Such aneurysms no doubt press on the upper surface of the cavernous sinus; just beneath is the ophthalmic division (*see Fig.* 199). There may then be very severe pain in the forehead, but there is no anæsthesia, although the pain can have a strictly anatomical distribution. Angiography demonstrates the peculiar flask-like shape of those aneurysms which spring from the intradural, supraclinoid part of the internal carotid artery (*Fig.* 215).

Syphilis is a not uncommon cause of ophthalmoplegia and is a possibility that must be borne in mind. Some of these cases might be aneurysmal.

In cases where the evolution of the cavernous syndrome is slow, it is not always easy to be sure of the cause. Aneurysm is still a possibility. Meningiomas in the middle fossa may involve the cavernous sinus, but they differ from aneurysms in frequently causing monocular amblyopia or blindness and not so commonly paralysing the nerves in the wall of the cavernous sinus. Aneurysms are apt to do the reverse. In one case a carcinoma arising in the glands in the roof of the naso-pharynx produced blindness in the right eye, abducens palsy, and anæsthesia of the right ophthalmic division of the trigeminus, by invasion of the sphenoid, cavernous sinus, and sella. In another, a man aged 68, there was progressive exophthalmos and blindness of the left eye over a period of three years, and finally intense pain and anæsthesia over the ophthalmic division. The sensory root of the trigeminus was cut under local anæsthesia, and at the same time a portion of the tumour

removed for histology. It proved to be a tough local growth, histologically difficult to place, but probably arising from a sinus.

In three cases oculomotor palsy has been produced by pituitary adenomata. They will be recorded elsewhere.

FIG. 215.—Angiogram of a case of supraclinoid aneurysm causing oculomotor palsy but no trigeminal palsy.

PATHOLOGY

Nothing has been said about the pathology of these aneurysms except that only one of them was even questionably syphilitic, which undoubtedly accounts for the relatively good prognosis of the others. They occur in persons in all the decades of life from the third onwards, in those who are to all appearances normal (though *Cases* 1 and 12 were arteriosclerotic, *Case* 5 had a very high blood-pressure). The studies of Eppinger, Turnbull, Drennan, Forbus, and Tuthill have demonstrated a tendency to congenital deficiencies in the walls of the cerebral vessels, but otherwise reveal nothing of particular interest in the structure of the sacs. It may be conjectured that the enclosure of the cerebral arterial supply within the bony skull has reduced the necessity for protection by means of the connective-tissue sheaths which are a feature of the vessels in other situations. This is well enough save when repair, when buffering from without to withstand intrinsic disease processes, is called for. John Hunter found that aneurysms of the limbs

were difficult to produce experimentally, because the outer fibrous coat is repaired by the neighbouring connective tissue. In the cavernous sinus the opportunities for such healing are at a minimum; local injury must much more easily lead to herniation of the vessel wall, for there is nothing that can come in contact with it until it has bulged out. There is no vascular sheath in the cavernous sinus which could help to make good a defect. The aneurysm, once it commences, must increase in size (if it does not burst into the cavernous sinus) until it comes in contact with the dura. Whilst it is a fortunate thing for the patient that this happens, security is bought at a price, because of the important structures which this particular stretch of dura chances to harbour. These points in clinical pathology are clear enough, but the really fundamental reason for the prevalence of aneurysms here is still obscure. It may be that the carotid artery, as soon as it enters the skull, loses some of its outer supporting coat and that the S-shaped bends which have been thought to protect the smaller vessels of the brain from the effects of sudden alterations in cardiac output may be a source of weakness. Another curious fact which deserves notice is the preponderance of females, which is so striking that it might be thought that the present experience is somewhat fortuitous. However, the peculiar vulnerability of the female to this type of non-syphilitic aneurysm has been brought to light by others (Fearnsides, Schmidt). There must be some message contained in that fact could we but decipher it. Any attempt to do so must lead us into endocrinal or other recondite speculation on the dangers of being female. It may be recalled that in both Travers' case (1810) and Dalrymple's (1815), the first arteriovenous cavernous fistulæ to be successfully treated surgically, both the women were pregnant at the moment of onset. Neither case was traumatic.

In the literature many cases will be found in which the aneurysm was discovered at necropsy on a patient who died in an insane asylum. Many of the beautiful specimens in the Royal College of Surgeons Museum are from such sources. The inference, so far as it concerns pathology, must surely be that massive intracranial aneurysms are prone to produce mental disorder, rather than that the insane are liable to aneurysm. In the present paper *Cases* 2 and 5 remind us vividly of this fact. Both became mentally disordered, but only after recognizable signs of aneurysm had made their appearance.

Role of Trauma.—The possibility of trauma causing a saccular, instead of an arteriovenous, aneurysm must be borne in mind. Admittedly the majority of traumatic aneurysms are of the communicating or fistulous variety, but they can be saccular. Conversely, we must not lose sight of the fact that a significantly high proportion of fistulous aneurysms are non-traumatic (about 25 per cent according to C. H. Sattler). It seems to the writer that the fistulous aneurysms of so-called 'spontaneous' development are fundamentally the same as those that have been here described. The difference, therefore, between the non-traumatic saccular variety and the non-traumatic fistulous must be a fortuitous one, a matter of degree, very different though the final clinical characters of the two types will be. If a weak point develops on the carotid wall and bursts into the cavernous sinus before it has attained any size, the average picture of a pure fistulous aneurysm will ensue. If, on the other hand, the weak point allows the formation of a considerable sac, or if the whole vessel enlarges in a fusiform manner, the aneurysmal wall must come into contact with the dura of the cavernous sinus. From this it secures such extramural support that its further progress is retarded. Its pressure on the branches

of the trigeminus contained in the wall of the sinus leads to the agonizing pain and facial anæsthesia so characteristic of the aneurysmal syndrome. Furthermore, its pressure on the motor nerves to the eyeball induces those widespread ocular palsies which complete the picture. The usual absence of these local paralyses in the fistulous aneurysms is evidence that the aneurysm bursts when small or little more than a weak point in the arterial wall. Clearly this damaged focus might be the result of injury, though commonly it is not so. However, one of Byrom Bramwell's non-fistulous aneurysmal cases that ended in an asylum, there to die, had had a severe head injury fifteen years before. Kirby's patient had had a bad head injury thirty-four years previously, Peacock's and Zollinger and Cutler's eight years before. The subject is further reviewed by Borchardt.

The whole question of trauma may be summed up by the statement that injury may damage the coats of the carotid artery, and lead to an aneurysm which is usually fistulous, but sometimes saccular; that an aneurysm may develop from non-traumatic causes, and that such a one is commonly saccular, but can be fistulous. It should be added that the difference between fistulous and non-fistulous deformations probably depends on the extent of the damaged area, small in the former, larger in the latter. And, maybe, a last addendum should be made for the sake of clarity. It has been assumed by me that the fistulous cases go through an aneurysmal stage (as happens in secondary hæmorrhage—and *see* Dalrymple's case). It is not actually known how often this is the case, but it cannot be accepted that the alternative is the uniform manner of evolution, i.e., an instantaneous but controlled tear through all the coats of the artery putting its interior into immediate communication with the venous blood of the cavernous sinus.

RADIOLOGICAL APPEARANCES

X-ray evidence of the presence of an aneurysm was found in 10 cases out of the 16. The most convincing proof that one can have is the visualization of the calcified wall of the aneurysm. The time factor is important here, and several years are necessary for lime salts to be deposited in great enough quantity for them to throw a shadow. In *Case* 2 the whole shell was sufficiently calcified after five years to produce a faintly opaque, inferiorly truncated, globular shadow in the floor of the middle fossa. It is only when exceptionally dense that the calcium deposits in the whole circumference cause the aneurysm to cast a uniform shadow. In *Case* 1 no shadow was visible after the same length of time, and it was not actually discovered till ten years after the date of presumed onset. During the next seven years it increased very slowly in sharpness. These differences suggest that individual metabolic variations enter into this question of calcification, and it follows that, whereas one may see the aneurysmal wall defined in the radiograph (sometimes within three years), one cannot expect to find it invariably, or to lay down any rule as to when it should appear. In *Case* 16 shadows were present when the first unequivocal signs of aneurysm made their appearance, indicating that it must already have been present for a long time. The calcification in the infraclinoid aneurysms is parasellar, and usually shows up as a more or less regular and crisp line, narrow but dense—a true picture of a section of the cortex of the aneurysm. The cortical shadows have been described by Sosman and Vogt and by Albl, and are sometimes, and unfortunately, spoken of as Albl's rings, a term

that has no special meaning. The paper of Sosman and Vogt, as has been said, is a most important contribution, because they describe from the radiological viewpoint the whole of the Brigham Hospital material.

The parasellar situation of the aneurysm eventually leads to a certain amount of sellar deformation, but to the expert eye there are rarely any difficulties in distinguishing it from that of an expanding intrasellar tumour. Mention has been made of the fact that a pituitary tumour can, very rarely, cause a cavernous sinus syndrome by breaking through its adenomatous capsule laterally, and on radiological examination alone it may be impossible to differentiate the cause.

Two points in radiographic diagnosis still remain to be dealt with: the search for changes in the sphenoid at the apex of the orbit (in other words at the anterior end of the cavernous sinus), and angiography. Studies of the orbit by axial views to show the optic canals have been used in all cases since the quality of the information obtainable by that means was discovered by E. W. Twining and the author. Two important structural changes can be brought out in this way—enlargement of the superior orbital fissure, and erosion of the lower and outer wall of the optic canal; examples have already been given, both in this paper and in others.[38, 39] Most aneurysms are anteriorly placed in the cavernous sinus and exert pressure upon the sphenoidal wing. The anterior end of the S-bend of the carotid ordinarily grooves the sphenoid as the vessel curves up to pierce the dura medial to the anterior clinoid process. Aneurysmal herniation here is certain in time to porose the bone, and to wedge itself into the orbital fissure. It may come to elevate the lesser sphenoidal wing and clinoid process (as it did in *Case* 2, and *see Fig.* 213), and to wear away that part of the greater wing which forms the lower boundary of the sphenoidal fissure. At the same time it compresses the body of the sphenoid and the strut of bone, the posterior root of the lesser sphenoidal wing, which passes from there to the lesser wing to complete the optic canal. In only one case has this wall entirely disappeared (*Fig.* 216). In only one case (*Case* 2) in the present series has a defect of vision been observed (save cataract) other than the disability produced by loss of the power of accommodation from paralysis of the third nerve. Another view may be instructive; a basal projection may disclose considerable enlargement of the foramen lacerum medium, through which the carotid artery enters the skull. Such a change can only be demonstrated in very large aneurysms, and was seen in the present series only in *Cases* 2 and 4 (*Fig.* 217). It will be looked for especially in *Group A* of the cavernous syndromes, where a considerable length of artery is expanded away back to its entrance into the skull. Dr. Twining's own studies will appear shortly.

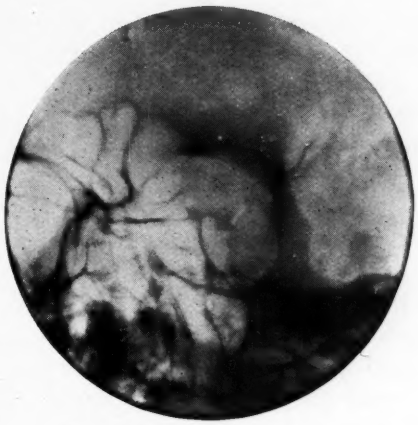

FIG. 216.—Destruction of lower wall of optic foramen in a cavernous aneurysm.

Angiography.—The injection of thorotrast into the carotid artery, a method introduced by Egas Moniz as a means of localizing intracranial tumours, can be most useful in aneurysms. Examples have been brought forward in this paper.

The injections were made, under omnopon-scopolamine-novocain anæsthesia, directly into the common or internal carotid vessels. Much less thorotrast is needed if the injection is made into the internal carotid—7 or 8 c.c. as against 16 to 20 c.c. Lima's needle may be used, or an ordinary Record needle can be conveniently bent. It is important to use a needle mounted on a short rubber

FIG. 217.—*Case* 3. Basal projection showing erosion of petrous tip on left side; enlargement of carotid canal.

connecting piece, so that movements of the neck do not disturb or damage the artery. Even though there is an aneurysm in the skull, the cervical carotid trunks are usually perfectly supple and healthy, and bear puncture quite safely. Lateral films are best taken with the patient supine and face upwards, with the rays shot through from the side, a Lysholm grid being used. The information given by angiography is useful in that the diagnosis is confirmed as a rule, but the size of the aneurysm may seem to be much less than one would have imagined. The aneurysm shows up as an opaque area covering the sella turcica. Most aneurysms are filled to some extent with laminated clot, some with much, some with less. The

scour of blood and thorotrast within the cavity varies, therefore, considerably in size and can be disappointingly small. If it is very large the indication to tie the carotid will be more definite. The cavernous aneurysms show up as a rule very well, for they are offshoots of the main trunk. A supraclinoid aneurysm is easily distinguished from the subclinoid. The former presents a pyriform appearance a centimetre or so long, with its long axis directed backwards. It is usually bottle-necked and its rounded end is not commonly very much wider than its stalk. A very typical appearance is that shown in *Fig.* 215. Aneurysms on the more distal branches may not be visualized, as was proved in one case. In *Case* 12 a relatively poor picture was obtained. (The reader is referred further to an important paper by Dott and his co-workers that appeared after this paper was written.)

TREATMENT

One cannot approach the subject of ligature for the saccular type of aneurysm without thoughts of the treatment of the fistulous variety, and indeed without comment on carotid ligature in general. With arteriovenous aneurysms the problem is how to cut down the blood-supply sufficiently, for ligature of the carotids in various combinations (common and internal or external; both internal and external; internal or common alone) often fails to bring about a cure (success in no more than 40 to 50 per cent according to Dandy's recent estimation). The whole problem has been very fully discussed by that surgeon. He has put a silver clip on the internal carotid intracranially in two cases where cervical carotid ligature had failed, securing the vessel a second time at the point where it pierces the dura—a brilliant conception successfully carried out. The necessity for such a step as this came from the heavy contribution to the aneurysm which came from above, through the circle of Willis.

In the saccular type anxiety is of a different order, for now there are fears that the circulation of the brain will be cut down too much if even the common, let alone the internal, carotid is tied. The view of Delbet, that the collateral circulation is likely to be more efficient if an aneurysm is present because there is already an impediment to the direct circulation which should have facilitated the development of collaterals, has received a certain measure of acceptance. However true Delbet's view may be so far as concerns the circulation of the limbs, there is evidence that an aneurysm might act in the opposite direction inside the skull. Thus hemiplegia and death followed a partial occlusion of the internal carotid at Dandy's hands in a man aged 33. The aneurysmal sac (from the posterior communicating artery) was found at necropsy to have compressed the middle cerebral artery and impeded the circulation. Although paresis did not appear until seven hours after ligature, no thrombosis was found. The brain was said to be simply ischæmic, a curious fact when we recall that the cortex becomes unexcitable after ten minutes of complete anæmia. This case demonstrates how important it is to make angiographic studies of the aneurysms, and not to ligature the main artery unless the aneurysm is shown to spring directly from its trunk.

Experiences such as that just cited make the surgeon chary of tying the carotid, and it is likely that the risks will always remain individual and idiosyncratic to each case. The effect of immediate ischæmia can be tested by trial digital compressions, and by test ligatures under local anæsthesia. The ill result that cannot be avoided,

because it cannot certainly be foreseen, is the late paralysis. Perthes (1920) drew attention to this type of accident, believing it to be due to embolism of the cerebral vessels from clot formed at the site of ligature. It might, therefore, occur more often after common carotid ligature than after that of the internal, because the blood-flow from the external might sweep clot onwards into the cranium. This is, most probably, untrue. Bruning (1926) observed that some of the late paralyses recovered spontaneously, and suggested that the untoward sequelæ could be subdivided into: (*a*) Primary cerebral anæmia because of a faulty development of the circle of Willis (prognosis bad); (*b*) Primary arterial thrombosis or embolism (prognosis bad); (*c*) Venous thrombosis, probably through slowing of the circulation, and local œdema (prognosis good).

Case 15 of the present series illustrates the anxieties that may arise in these cases. One cannot know what the lesion is in the recoveries; Bruning's suggestions of œdema and venous thrombosis is as good as any, and was probably the cause of the fortunately transient paresis and aphasia in this case. It is quite certain that the effects of ligature require reassessment, and that new observations on the exact pathological cause of failure are badly needed. It may be that the bad reputation which carotid ligature bears is less well deserved for such cases as cavernous carotid aneurysm, whether fistulous or not, than for other conditions. Siegrist thought that in only 16 out of 371 fatalities after ligature of the common carotid artery could circulatory failure be regarded as the sole cause of death. This conclusion should be accepted with reserve. The present writer has tied the internal or common carotid for aneurysm 12 times, and there have been 2 fatalities, 1 transient paresis, and 9 successes. The two failures were in middle-aged women extremely ill from leaking intradural carotid aneurysms. In such cases the raised intracranial pressure from the effused blood, a rise due to the mass of free blood plus the osmotic attraction of cerebrospinal fluid, already hampers the circulation to a degree that renders immediate ligature unwise. These two patients would probably have died had they been left alone. There seems to be justification for Trotter's belief that bad results are much more apt to occur if the carotid wall and sheath are abnormal. The ill repute of ligature of the carotid vessels has grown out of statistics compiled from cases of infection, of secondary hæmorrhage, of malignant disease of the neck, and so forth. We need new statistics on the subject.

In all cases preliminary compression of the artery should be carried out. At the actual operation, which will be done under local anæsthesia, the artery must first be tied gently with catgut. A piece of fascia under the knot allows it to be cut safely and quickly if need be. Dynamometer readings of the grip are then taken every ten minutes during the next hour, and if all is well a waxed silk ligature is applied, without removing the temporary tie, and the wound sutured. This has been the method employed in my own cases. The only case in my experience in which compression proved that the case was unsuitable for ligature was a bad arteriovenous fistula. This patient began to nod, to go dizzy, to speak indistinctly, and to lose power down the right side of his body within a minute of digital compression of the left common carotid artery. Believing that the result might be due to stimulation of the carotid sinus or vagus in unselective pressure of this kind, the arteries were exposed and a temporary ligature placed on the internal carotid, with precisely the same result; no other course was open but to remove the ligature and leave the patient alone. In this case there must have

been a bad deficiency in the circle of Willis. It is unwise to reopen the wound and manipulate the vessel again until it is firmly cicatrized. Thus, following a thorotrast injection, the vessel should be tied at once or not for some weeks, otherwise the case comes under the heading of one with an abnormal arterial wall. Half closure with a metal band with full closure in a day or so seems to be equally open to objection, though Matas has made successful use of this procedure. That preparation by periodic compression improves the circulation was demonstrated by one of Matas's cases in which the band had to be removed four and a quarter hours after it was applied because of deepening coma. After five weeks of episodic digital compression the artery was occluded again, this time without ill effect. A period of preparation, the exact length of which is unknown, is advisable.

Which artery should be tied, the internal or the common carotid? There is no doubt that ligature of the internal carotid cuts off the blood-supply most thoroughly. If the common carotid alone is tied the collateral circulation through the external carotid is so free that blood will eventually find its way again into the skull. Ligature of the common carotid for hæmorrhage is of little use, but the needs in aneurysmal cases are different. It will be remembered that after ligature of the main artery to a limb, to use a parallel case to help in clarifying the problem, the pulse very rarely fully returns, although the extremity survives. This means a cutting down of the intravascular tension, and it might be enough to favour thrombosis in the aneurysmal sac, a condition towards which there is a strong natural tendency, even in untreated specimens.

In older people, therefore, ligature of the common carotid may well suffice. The practice of adding to this ligature the tying off of the more accessible of the branches of the external carotid, as was done in one of my own cases, may also commend itself to others. The advantages of this method is that it reduces the possibilities of the free collateral circulation which can decrease the virtue of a common carotid ligation.

The necessity for arterial ligature is not quite so imperative in the case of the saccular aneurysm as it is in the arteriovenous, where progressive deterioration is certain. Each case should be treated on its individual merits, having regard to the age, character, and life expectation of the patient. On these grounds the aged arteriosclerotic patient will be left alone, for an early exitus is by no means certain even if nothing is done. The special indication for ligature is evidence, usually in the form of unremitting pain, that the aneurysm is not thrombosed but actively increasing in size. Once the patient has reached the state of *Cases* 2 and 4 nothing can be hoped for; had they been diagnosed early enough, their final disintegration might have been prevented by ligature. Some of the patients have not been operated upon at all because their lesion was stabilized, and because they were wlll satisfied with their state and not reduced to invalidism. Reflection on these cases brings out the importance of early diagnosis. Possibly some sufferers are doomed from the start, do what we will, but frankly we do not know yet if that be so or not. Most of these patients went through weeks and months of misery in their own homes or in hospitals with the cause of their distress unrecognized. It can be laid down as a principle that prompt ligature should be made unless there are such obvious objections as will make exception to the rule the wiser choice.

Exploration of the actual aneurysmal site needs no further comment here. Two aneurysms in the literature, besides the first case in this series, were discovered

at operation for neuralgia (Magnus, Harvey Cushing). Others have been operated on as tumours, abscesses, or clots (Reinhardt, Elsberg, Borchardt, Foerster, Trevani, Zollinger and Cutler, amongst others). Such experiences may yet befall others.

CONCLUSION

The intracavernous, subclinoid, saccular aneurysms of the internal carotid produce clearly recognizable syndromes, the investigational interest of which, by various means, clinical and radiographical, is only matched by the fascinating problem of their treatment, towards which we are now, after a conservative, and what maybe seems in the retrospect a fumbling, approach, crisply going forward. These cases combine two fascinating subjects—the surgery of the nervous system and of the arteries. We know to-day a certain amount about both, and we shall learn much more of the therapeutic possibilities when the cases are promptly recognized, giving the surgeon the opportunity of applying at an early stage that treatment which seems best suited to the individual.

BIBLIOGRAPHY

ADAMS, J., "Aneurisms of the Internal Carotid in the Cavernous Sinus", *Lancet*, 1869, ii, 768.

ALBL, H., "Aneurysma der Carotis Interna, einen Hypophysentumor vortauschend; ein Beitrag zur Diagnose intrakranieller Aneurysmen", *Fortschr. a. d. Geb. d. Roentgenstrahlen*, 1929, xxxix, 890.

ALBRIGHT, F., "The Syndrome produced by Aneurysm at or near the Junction of the Internal Carotid and the Circle of Willis", *Bull. Johns Hopkins Hosp.*, 1929, xliv, 215.

BARTHOLOW, R., "Aneurisms of the Arteries at the Base of the Brain: Their Symptomatology, Diagnosis and Treatment", *Amer. Jour. Med. Sci.*, 1872, lxiv, 373.

BEADLES, C. F., "Aneurisms of the Larger Cerebral Arteries", *Brain*, 1907, xxx, Oct., 285.

BIRLEY, S. L., and TROTTER, W., "Traumatic Aneurysm of the Intracranial Portion of the Internal Carotid Artery", *Brain*, 1928, li, 184.

BLANE, G., "History of some Cases of Diseases of the Brain, etc.", *Trans. Soc. Improvement of Med. and Chir. Knowledge*, London, 1800, ii, 192.

BORCHARDT, M., "Die chirurgische Bedeutung der Gehirnaneurysmen", *Beitr. z. klin. Chir.*, 1925, cxxxiii, 429.

BRAMWELL, BYROM, "Intracranial Aneurysms", *Clinical Studies*, 1906, 288. Edinburgh.

BRAMWELL, EDWIN, "A Note upon the Aetiology of Recurrent and Periodic Ocular Palsy and Ophthalmoplegic Migraine", *Trans. Ophthalmol. Soc.*, 1934, liv, 205.

BRÜNING, F., "Zur Chirurgie der Art. carotis", *Deut. med. Woch.*, 1926, lii, 145.

CUSHING, H., "Contributions to the Study of Intracranial Aneurysms", *Guy's Hosp. Rep.*, 1923, lxxiii, 159.

CZERMAK, "Verein deutscher Aerzte in Prag", *Prag. Med. Woch*, 1902, xxvii, 227.

DANDY, W. E., "Carotid-Cavernous Aneurysms (Pulsating Exophthalmos)", *Zentralb. f. Neurochir.*, 1937, ii, 77.

DANDY, W. E., "Intracranial Aneurysm of the Internal Carotid Artery", *Ann. of Surg.*, 1938, cvii, 654.

DAVIS, L. E., "Lesions of the Paratrigeminal Area", *Jour. Amer. Med. Assoc.*, 1923, lxxx, Feb., 830.

DALRYMPLE, W., "A Case of Aneurysm by Anastomosis in the Orbit cured by Tying the Common Trunk of the Left Carotid Artery", *Med.-Chir. Trans. Lond.*, 1815, vi, 111.

DELPECH, M., "Tumeur anévrysmale de la Base du Cerveau, etc.", *Bull. de la Soc. Anat. Paris*, 1845, xvii, 112.

DOTT, N., "Intracranial Aneurysms", *Edin. Med. Jour.*, 1933, Dec., 219.

DRENNAN, A. M., "Aneurysms of the Larger Cerebral Vessels", *N.Z. Med. Jour.*, 1921.

DURAND, *Des Anévrysmes du Cerveau, etc,*, 1868. Paris.

EPPINGER, H., "Pathogenesis der Aneurysmen einschliesslich des Aneurysma Equi verminosum", *Arch. f. klin. Chir.*, 1887, xxxv, suppl.
FEARNSIDES, E. G., "Intracranial Aneurysms", *Brain*, 1916, xxxix, 224.
FOGGIE, W. E., "Aneurysms of the Internal Carotid in the Cavernous Sinus", *Edin. Med. Jour.*, 1927, 649.
FORBUS, WILEY, "On the Origin of Miliary Aneurysms of the Superficial Cerebral Arteries", *Johns Hopkins Hosp. Rep.*, 1930, xlvii, 239.
FORD, F. R., and WOODHALL, B., "Phenomena due to Misdirection of Regenerating Fibres of Cranial, Spinal, and Autonomic Nerves", *Arch. of Surg.*, 1938, xxxvi, 480.
FOIX, C., "Syndrome de la Paroi externe du Sinus caverneux", *Rev. neurol.*, 1922, xxxvii, xxxviii, 827.
GARVEY, P. H., "Aneurysms of the Circle of Willis", *Arch. of Ophthalmol.*, 1934, xi, June, 1032.
GIRAUDET, E., "Observations sur les Anévrismes intracraniens", *Gaz. des. Hôp.*, 1857, 105.
GOLLUB, H., "Das Problem der Hirnstörungen nach Unterbindung der Arteria carotis communis", *Münch. med. Woch.*, 1936, lxxxiii, 1827. ..
GOUGUENHEIM, A., *Des Tumeurs anévrysmales du Cerveau*, 1866. Paris.
GULL, W., "Cases of Aneurism of the Cerebral Vessels", *Guy's Hosp. Rep.*, 1859, v, 281.
GUIBERT, M., "Anévrysme artériel de la Carotide interne au Niveau du Sinus caverneux gauche", *Ann. d'Oculist.*, 1895, cxiii, 314.
HERMANN, K., OBRADOR, S., and DOTT, N., "Intracranial Aneurysms and Allied Clinical Syndromes", *Lisboa méd.*, 1937, xiv, 782.
HIRSCHFELD, L., "Epanchement de Sang dans le Sinus caverneux du Côté gauche diagnostiqué pendant la Vie, *Gaz. des Hôp.*, 1859, xxxii, 57.
HOLMES, TIMOTHY, "Aneurisms of the Internal Carotid in the Cavernous Sinus", *Trans. Pathol. Soc. Lond.* 1860–61, xii, 61.
HOMANS, J., "Accidents and Precautions in Ligature of the Common Carotid Artery", *Ann. of Surg.*, 1920, lxxi, 707.
HUTCHINSON, J., "Aneurism of the Internal Carotid within the Skull", *Trans. Clin. Soc. Lond.*, 1875, viii, 127.
JEFFERSON, G., "Compressions of the Chiasma, Optic Nerves, and Optic Tracts by Intracranial Aneurysms", *Brain*, 1937, lx, pt. 4, 444.
JEFFERSON, G., "The Optic Canals", *Proc. Roy. Soc. Med.* (Sect. Radiol.), 1936, July, xxix, 47.
LEBERT, H., "Ueber die Aneurysmen der Hirn-arterien", *Berlin klin. Woch.*, 1866, iii, 209, 229, etc.
LOCKE, C. E., "Intracranial Arterio-venous Aneurism or Pulsating Exophthalmos", *Ann. of Surg.*, 1924, lxxx, 1.
MAGNUS, V., "Aneurysm of the Internal Carotid Artery", *Jour. Amer. Med. Assoc.*, 1927, May, lxxxviii, 1712.
MAHONEY, W., and SHEEHAN, D., "Experimental Ptosis in Primates", *Arch. of Neurol. and Psychiat.*, 1936, xxxv, 99.
MATAS, R., "Testing the Efficiency of the Collateral Circulation as a Preliminary to the Occlusion of the Great Surgical Arteries", *Jour. Amer. Med. Assoc.*, 1914, lxiii, 1441.
MATAS, R., "Discussion of Treatment of Carotid Cavernous Arteriovenous Aneurysms", *Ann. of Surg.*, 1935, cii, 920. (*See also* SHIPLEY, WINSLOW, and WALKER.)
MATAS, R., "Aneurysms of the Circle of Willis", *Ann. of Surg.*, 1938, cvii, 660.
MCKENDREE, C. A., and DOSHAY, L. J., "Visual Disturbances of Obscure Etiology produced by Focal Intracranial Lesions Implicating the Optic Nerve", *Bull. Neurol. Inst. N.Y.*, 1936, v, 223.
MCKINNEY, J. McD., ACREE, T., and SOLTZ, S. E., "Syndrome of Unruptured Aneurysm of the Intracranial Portion of the Internal Carotid Artery", *Bull. Neurol. Inst. N.Y.*, 1936, v, 247.
NATTRASS, F. J., "Aneurysm of the Carotid Artery in the Cavernous Sinus", *Edin. Med. Jour.*, 1928, Jan. 30.
OLLOZ, M., "Ueber totale einseitige Ophthalmoplegie. Ein Fall von Aneurysma der Carotis interna im Sinus cavernosus mit Foix'schen Syndrome", *Schweiz. Arch. f. Neurol.* 1935, xxxv, 123.
PARKER, H. L., "Aneurysms of Cerebral Vessels", *Arch. of Neurol. and Psychiat.*, 1926, xvi, 728.
PEACOCK, T. B., "Intracranial Aneurysm", *St. Thomas's Hosp. Rep.*, 1876, 119, 317.
PERTHES, "Ueber die Ursache der Hirnstörungen nach Karotisunterbindung und über Arterienunterbindung ohne Schädigung der Intima", *Arch. f. klin. Chir.*, 1920, cxiv, 403.

PFINGST, A. O., and SPURLING, G., "The Role of Intracranial Aneurysms in the Production of Ocular Palsies", *Trans. Amer. Ophthalmol. Soc.*, 1929, xxvii, 227.
PITT, N., "The Goulstonian Lectures on Some Cerebral Lesions", *Brit. Med. Jour.*, 1890, i, 827.
REINHARDT, A., "Ueber Hirnarterienaneurysmen und ihre Folgen", *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1913, xxvi, 432.
ROMBERG, quoted by WILDBRAND and SAENGER.
SATTLER, C. H., "Pulsierender Exophthalmos", *Handbuch der gesamten Augenheilkunde*, 1920. Berlin.
SCHMIDT, M., "Intracranial Aneurysms", *Brain*, 1930, liii, 489.
SHAW, B. H., "Case of Double Sacculated Intracranial Aneurysm", *Jour. of Ment. Sci.*, 1901, xlvii, 547.
SHIPLEY, A. M., WINSLOW, N., and WALKER, W. W., "Aneurysm in the Cervical Portion of the Internal Carotid Artery", *Ann. of Surg.*, 1937, cv, 673.
SIEGRIST, A., "Die Gefahren des Ligatur der grossen Halsschlagadern für das Auge, etc.", *Arch. f. Ophthalmol.*, 1900, l, 511.
SJØQVIST, O., Ueber intrakranielle Aneurysmen der Arteria carotis und deren Beziehung zur ophthalmoplegischen Migräne", *Der Nervenarzt*, 1936, ix, 233.
SMITH, R. W., "Multilocular Aneurysm of the Base of the Brain", *Proc. Pathol. Soc. Dublin*, 1842, i, 205.
SOSMAN, M. C., and VOGT, E. C., "Aneurysms of the Internal Carotid Artery and the Circle of Willis from a Roentgenological Viewpoint", *Amer. Jour. Roentgenol.*, 1926, xv, 122.
STOPFORD, J. S. B., "The Arteries of the Pons and Medulla Oblongata", *Jour. of Anat.*, 1917, xii, 267.
SYMONDS, C. P., "Contributions to the Clinical Study of Intracranial Aneurysms", *Guy's Hosp. Rep.*, 1923, lxxiii, 139.
TRAVERS, B., "A Case of Aneurism by Anastomosis in the Orbit, Cured by Ligature of the Common Carotid Artery", *Med.-Chir. Trans. Lond.*, 1810, ii, 1.
TREVANI, E., "Ein als parasellarer Tumor operiertes Aneurysma der Arteria Carotis interna", *Deut. Zeits. f. Chir.*, 1932, ccxxxvii, 534.
TROTTER, W., private communication.
TÖNNIS, W., "Erfolgreiche Behandlung eines Aneurysma der Art. commun. ant. cerebri", *Zentralb. f. Neurochir.*, 1936, i, 39.
TURNBULL, H. M., "Alterations in Arterial Structure and their Relation to Syphilis", *Quart. Jour. Med.*, 1915, viii, 201.
TUTHILL, C. R., "Cerebral Aneurysms", *Arch. of Pathol.*, 1933, xvi, 630.
DE VET, A. C., and ZECKEL, A., "Aneurysma van de Arteria Carotis interna", *Nederl. Tijds. v. Geneesk.*, 1937, lxxxi, 4145.
VIETS, H., "Unilateral Ophthalmoplegia—Report of a Case due to Carotid Aneurysm", *Jour. Nerv. and Ment. Dis.*, 1918, xlvii, 249.
VINCENT, C., THIÉBAUT, F., LEMOYNE, J., and GUILLAUMAT, L., "Deux Cas d'Anévrysme artériel intracranien traité par Ligature de la Carotide primitive", *Rev. neurol.*, 1937, lxvii, 361.
WILDBRAND, H., and SAENGER, A., *Die Neurologie des Auges*, 1915, ii. Wiesbaden.
WOOLF, E., *The Anatomy of the Eye and Orbit*, 1933. London.
ZOLLINGER, R., and CUTLER, E. C., "Aneurysm of the Internal Carotid Artery", *Arch. of Neurol. and Psychiat.*, 1933, xxx, 607.

CARCINOID TUMOURS OF THE APPENDIX

By THOMAS MOORE, Manchester

HISTORY

Most of the early cases of carcinoma of the appendix reported were undoubtedly examples of metastases spread transcœlomically from some primary intraperitoneal site. Thus, although the earliest report was made by Merling[1] in 1838, and during the next forty years Prus,[2] Rokitansky,[3] Kolaczek,[4] Leichtenstern,[5] and Bierhoff[6] recorded examples, Beger[7] in 1882 would appear to have reported the first case of primary appendicular carcinoma. In 1903 Elting[8] collected 40 cases from the literature. He thought that the evidence was only sufficiently strong in 20 of these to be reasonably certain that they had arisen primarily in the appendix. Of the 3 cases reported by Moschcowitz[9] in the same year 2 undoubtedly originated in the appendix. Rolleston and Jones[10] in 1906 wrote a very instructive paper on the 42 cases they were able to find in the literature.

These early writers stressed the association of the disease with "obliterative appendicitis", and noted that these tumours did not infiltrate widely or give rise to metastases in the lymphatic glands. In spite of this, however, no suspicion was aroused that these neoplasms were not ordinary adenocarcinomata. Some interesting tumours of the small intestine were also being encountered about the same time. In 1888 Lubarsch[11] published an account of 2 very interesting cases, in which two and six small separate tumours, respectively, were found in the submucosa of the terminal ileum. These microscopically were seen to consist of columns and masses of cells which were not like those of the ordinary carcinomata. There was no true glandular formation, although degeneration of some of the central cells sometimes gave a pseudo-glandular appearance. Although he was able to trace the connection of some of these cells with the crypts of Lieberkühn, he had no doubt that these tumours were not the usual type of adenocarcinoma. Nevertheless he labelled his cases "primary carcinomata of the ileum". Bunting[12] in 1904 reported a case of the common type of adenocarcinoma of the ileum and one of the variety described by Lubarsch.

It was not until 1907 that Oberndorfer[13] clarified the position. He showed that in the gastro-intestinal tract there occurred, in addition to the ordinary adenocarcinomata, a superficially related type of tumour, for which he coined the term "carcinoids". The distinguishing features of these he believed to be: their localized nature and apparent benignity as shown by their slow rate of growth and lack of infiltration; the absence of metastatic spread by lymph and vascular channels; the fact that more than one tumour was often present; and their characteristic histological appearance.

Oberndorfer's idea was generally accepted, and numerous papers appearing after this were concerned not with the existence of these tumours, but with their probable site of origin. In this country, however, they continued to be classified as carcinomata. In 1913 Graham[14] reported 6 cases. From his account they all

appear to have been examples of carcinoid tumours. He called them, however, spheroidal-cell carcinomata, and made no reference to Oberndorfer's writings. Eight years later Wilkie[15] published 3 beautiful cases of carcinoids of the appendix, but presented them as spheroidal-cell carcinomata.

PATHOLOGY

It is now well recognized that carcinoids may occur anywhere in the gastro-intestinal tract from the cardia to the lower end of the rectum. They are found most commonly in the appendix. Illingworth and Dick[16] believe they constitute 0·4 per cent of appendicular lesions. They are by far the commonest neoplasm encountered here, constituting about 90 per cent of the total. Reimann[17] found 17 appendicular neoplasms in 13,151 appendices removed at operation. Fourteen of these were carcinoid tumours and 3 adenocarcinomata. A single tumour is usually present, although elsewhere in the intestine multiplicity is quite common. In 1916 Simon[18] could only find 5 reported examples of multiple appendicular carcinoids.

A nodule of characteristic yellow colour, and of varying size, is found in the submucosa. Usually the mucous membrane is intact over it, although on occasion some superficial ulceration may have occurred. It is of a firm rubbery consistency, and sometimes may extend through the muscle to involve the peritoneal coat. The cut surface of the tumour often shows fibrous strands dividing it into separate yellow nodules. As it enlarges the tumour may come into contact with the opposite wall of the appendix and cause obstruction of the lumen. On other occasions it occurs as an annular thickening of the appendix wall sometimes as much as half an inch in length, which completely obliterates the lumen. In either event an attack of acute obstructive appendicitis may supervene. It is interesting to note that the tumour obliterates the lumen by its bulk and not by any associated fibrous contraction. The lesion may be so small that it is only found on microscopical examination.

The tip of the appendix is most commonly affected. Simon in 139 cases found 77 per cent occurred in the distal third, 14 per cent in the middle third, 7 per cent at the base, and 2 per cent involving the whole organ. MacCartey and McGrath[19] found 70 per cent of their 40 cases occurred at the tip. Of the 17 cases reported by Raiford,[20] 12 were situated at the tip, 3 in the middle, and 2 at the base. According to numerous statistical reports the condition is three times more common in the female than in the male; 70 per cent of the cases found at operation occurred below 30 years of age. In Oberndorfer's series[13] the average age-incidence of operation cases was 24 years of age, while for cases found at autopsy it was 57 years of age.

Although the main feature of the tumour as originally described was its benignity, it is now recognized that occasionally a malignant change may occur. Spread takes place by continuity and contiguity of tissue as well as by lymph and vascular channels. In the small intestine such a change is not infrequently encountered. Cooke[21] in 1931 found that just over 20 per cent of all reported small intestine carcinoids were malignant. Metastases were found in the peritoneum, lymph-glands, and liver. Appendicular carcinoids are very rarely malignant. In 1926 Stewart and Taylor[22] reported a case, and could find only 8 examples in the literature. Wyatt[23] recently has been able to collect another 5 cases. Metastases are found only

in the lymph-glands and peritoneum; spread by the blood-stream is extremely rare. The only case of this I have been able to find in the literature is that of Callender, quoted by Shaw,[24] in which metastases were found in the liver.

Microscopically the tumour is seen to consist of clumps and masses of large spheroidal cells lying in a fibrous stroma in which there is sometimes a good proportion of involuntary muscle fibres. At the periphery of the tumour there is always some condensation of non-striped muscle fibres in the fibrous capsule which tends to form. The cells often penetrate beyond this, however, and numerous nests may be seen in the muscle or even the subserous coat.

The cells have round distinct nuclei, but in the larger cell masses the cytoplasmic borders are often ill defined. They bear no resemblance to the ordinary columnar or mucous cells lining the intestinal glands. There is no true gland formation. In the centre of some of the smaller cell masses there is sometimes a vacuole which contains an eosin-staining colloid material. The rosettes which occur in the neuroblastomata may be thus closely simulated.

The protoplasm of the cells contains much lipoid material, which accounts for the yellow colour of the tumour. The granules, which also occur in the cytoplasm, are stained yellow when fixed in chrome salts. They also give the argentaffin reaction described by Masson. This worker has also pointed out that three morphological types of cells can be distinguished in the carcinoid tumour: (1) Round or polygonal, the commonest cell, which makes up most of the cell masses; (2) Palisade cells, large and more oblong, occurring at the periphery of the cell masses—he likens these to the cells of the adrenal cortex; and (3) Columnar cells, forming the small rosettes already described. Although the argentaffin reaction described by Masson has done much to separate these tumours into a definite class they are quite easily recognized by their characteristic morphology even when stained by ordinary methods.

ORIGIN

There have been many interesting theories held as to the origin of carcinoid tumours. It was only natural that they should first be considered to arise from intestinal epithelium. In 1904 Bunting had suggested that they were carcinomata and corresponded to the basal-cell carcinomata of the skin. He later received great support from Burckhardt[25] in 1909 and Krompecher[26] in 1919. Oberndorfer rather favoured the idea of pancreatic intestinal rests as their site of origin. At that time these had been suggested by Trappe[27] to be the site of origin of intestinal adenomyomata. Toenniesson[28] in 1910 suggested that they arose from submucosal epithelial rests, thus falling into line with the cell-rest theory which was generally held at that time to account for the origin of most neoplasms. Shaw and numerous later writers also subscribed to this hypothesis. Saltykow,[29] owing to the similarity of their cells, suggested that they arose from the islet part only of pancreatic rests. Because of the yellow colour of these tumours Huebschmann[30] in 1910 thought they might arise from the "gelbe Zellen". This name had been applied to the many cells bearing yellow granules demonstrated in the crypts of Lieberkühn by Schidmt[31] in 1905. He believed them to be the same as those described by Kultschitzky[37] in 1887. In 1914 Gosset and Masson[32] made a complete study of the chromaffin tissue of the intestinal wall. They showed that such cells stained by

ammoniacal silver salts contained black granules in the protoplasm. By this method they were able to demonstrate such cells lying between the ordinary columnar cells near the bases of the crypts of Lieberkühn. Their studies of carcinoid tumours, using similar stains, demonstrated that they consisted of the same type of cells. They therefore re-named them argentaffin tumours. Hasewaga[33] in 1923, Danisch[34] in 1924, and Forbus[35] in 1925 confirmed Masson's work. Masson believed that these argentaffin cells (Kultschitzky) which are normally present in the intestine are of endodermal origin. On the other hand, however, Danisch, as the result of embryological studies, thought that these cells migrated to the bowel wall from the chromaffin tissue of the sympathetic ganglia and were therefore ectodermal in origin.

Later, Masson[36] published evidence that in partially obliterated appendices (chronic appendicitis) neuromata could be found in the small non-medullated nerve-plexuses in the submucosa. These always contained argentaffin cells, and, he asserted, retrogressed if the latter died. He postulated that these cells had reached this position by migration from the intestinal mucosa. From them he believed a carcinoid tumour might arise and spread first by intranervous paths. Later the surrounding connective tissue of the submucosa was involved. When this took place hypertrophy of muscle fibres normally present there occurred, and supplied the muscular tissue of the stroma. These changes, he assumed, were in response to the secretion of the carcinoid cells, which in normal conditions might exert a hormonal control over the muscularis mucosæ. In actual fact the muscle is found mainly at the periphery of the tumour and is not always a conspicuous feature of the true stroma. The internal secretion of these cells might be obtained from extracts of the carcinoid tumours.

No further knowledge of the carcinoid tumours has been published since this time. Although Masson's work is not accepted *in toto*, it is generally agreed that the Kultschitzky cells are the site of origin of these tumours. Thus they would be more accurately called the argentaffinomata, but the adjective "carcinoid" seems to be deeply rooted in medical nomenclature.

To account for their high incidence in the appendix it has been asserted that the Kultschitzky cells are more common here and in the terminal ileum than elsewhere in the intestine. The neoplasm grows very slowly, and in the intestine often must be missed at autopsy because of its relatively small size. In a small blind tube like the appendix it will more probably be noticed. Of more importance, however, is the likelihood of appendicular obstruction and consequent inflammatory changes by which attention may be drawn to it.

CLINICAL FEATURES

The disease has never been diagnosed before operation or autopsy. It may be responsible for the onset of an attack of acute obstructive appendicitis, or in cases without marked infection a mucocele may result. Although in a case of so-called chronic appendicitis a carcinoid may be present, no case has been reported where this has been diagnosed before operation. It is interesting to speculate as to the relationship of the neuromata described by Masson and the pain of chronic appendicitis, which in the absence of appendicular obstruction is at present inexplicable.

PROGNOSIS

There is very rarely any extension of the tumour. A few cases have been reported of spread to the lymphatic glands. Only one case with secondary deposits in the liver has been reported. The results of simple removal of the appendix are excellent, and the tumour is clinically benign.

TREATMENT

The presence of the tumour is usually discovered after appendicectomy has been performed, so that no special treatment is considered. Should, however, the lesion be diagnosed before this, simple appendicectomy will suffice. If extensive glandular involvement has occurred, this would necessitate a classical excision of the right colon with the involved glands. The prognosis after this is excellent.

AUTHOR'S CASES

Three years ago the author encountered within about six months two cases of acute obstructive appendicitis caused by carcinoid tumours. A search of the records of the Royal Victoria Infirmary, Newcastle-on-Tyne, to which he was then attached, was undertaken, and in the ten-year period 1927–36, 10 cases of appendicular carcinoids were found. During this time 10,229 appendicectomies were performed in the hospital. This incidence of 0·1 per cent is below that regarded as usual for carcinoids. The explanation undoubtedly is that in a large hospital only appendices showing specially interesting macroscopic features are further investigated. Thus numerous small carcinoids would be missed. Only one other appendicular neoplasm—a carcinoma—was encountered during this period. Carcinoids thus constituted 91 per cent of all the appendicular tumours found.

The youngest patient was 9 and the eldest 37 years of age. The average age was 24 years. There were 6 males and 4 females.

In 4 cases the tumour was situated at the tip of the appendix, in 5 in the middle third, and in 1 in the basal third. There were 9 cases pathologically benign, and 1 malignant. Of the 10 cases, 1 (the malignant case) died suddenly after a second operation for radical excision of the affected glands had been successfully performed; 2 cases died within the next few years from unrelated disease—at autopsy no recurrence could be found; and of the remaining 7 cases, 3 could not be traced, and the other 4 are alive and well, with no evidence of any recurrence at varying periods of at least one year after operation.

Relation of the Tumour to the Symptoms.—These cases may be classified into three groups according to the part played by the carcinoid in the causation of the symptoms:—

Group 1.—In 6 cases obstruction of the appendix had occurred due to the tumour, and an acute inflammatory change of varying intensity supervened (*Cases* 1–6).

Group 2.—In 3 cases the disease was associated with acute appendicitis, but had no aetiological significance (*Cases* 7–9).

Group 3.—In 1 case a small tumour was present in an otherwise normal appendix. It is probable from the clinical history that it played no part in the causation of the symptoms (*Case* 10).

CASE REPORTS

Case 1.—H. C., female, aged 23 years.

HISTORY.—Admitted on June 21, 1928, complaining of gradual onset of central abdominal pain two days before admission. For the last few hours this had decreased in severity, but had been more noticeable in the right iliac fossa. No previous attacks. No vomiting.

ON EXAMINATION.—General condition was good. Temperature 98·4 F. Pulse 104. There were tenderness and rigidity in the right iliac fossa; no mass.

OPERATION (June 21, 1928).—Rutherford Morison incision in right iliac fossa. The appendix was lying behind the cæcum and there were numerous enlarged glands in the ileo-cæcal angle which were thought to be tuberculous. Appendicectomy was performed and the wound closed. The patient made an uninterrupted recovery.

PATHOLOGY.—In the middle of the appendix there was obliteration of the lumen caused by an annular thickening of the wall about ¼ in. long. There was distension of the appendix beyond this, with acute inflammation of the mucous membrane. There was no perforation.

Microscopical examination of this thickening showed it to be a typical carcinoid tumour. Masses of the typical cells were seen occupying the submucosa, with some infiltration in the muscle coat.

PROGRESS.—Because of the microscopical report and the presence of the glands noted at operation, the patient was re-admitted to hospital and a further operation performed on July 20, 1928.

The old incision was re-opened and extended. There appeared to be some new glands in the ileocæcal angle. The terminal 12 in. of the ileum, the ascending colon, and the lateral few inches of the transverse colon were mobilized and excised, the bowel being divided by the cautery. The bowel ends were sutured and invaginated. A lateral anastomosis was performed between the transverse colon and the ileum. The wound was closed with a drain to the retroperitoneal space.

The patient made excellent progress, but died suddenly on July 30, 1928. The bowels had been moved naturally without pain the night before.

At autopsy no further evidence of the disease could be found, and no cause for the sudden death. Unfortunately I have been unable to trace the section of the excised glands.

Case 2.—E. D., female, aged 19 years.

HISTORY.—Admitted on July 28, 1928. The patient complained of sudden onset of pain in the right iliac fossa twelve hours before admission, which was getting easier. No vomiting. Several previous slight attacks in the last few years.

PREVIOUS HISTORY.—Rheumatic fever three years ago.

ON EXAMINATION.—General condition good. Normal pulse and temperature. Large hypertrophied heart with double mitral and (?) aortic diastolic murmur. Abdomen tender in right iliac fossa; no rigidity; no mass.

OPERATION (Aug. 8, 1928).—Local anæsthesia; McBurney incision. The appendix showed inflammatory changes and was hard. Appendicectomy was performed and the wound closed.

PATHOLOGY.—The appendix showed an annular thickening in the middle third, very like a growth. Some distension of the appendix distal to it, with very early inflammatory change.

Microscopical examination showed the appendix to be the seat of a carcinoid tumour.

PROGRESS.—The patient made an uninterrupted recovery. No reply was received to a recent questionaire.

Case 3.—J. B., male, aged 21 years.

HISTORY.—Admitted on Sept. 29, 1933. There was a fourteen-days' history of attacks of pain in the right iliac fossa, occasionally shooting across to the left side. Made him feel sick, but he had not vomited. Had been constipated for fourteen days: previously quite regular.

ON EXAMINATION.—The general condition good. Temperature 100·4° F. Pulse 90. Abdomen: tenderness and rigidity in right iliac fossa; no mass.

OPERATION (Sept. 29, 1933).—Rutherford Morison incision in right iliac fossa. Very large adherent retrocæcal appendix removed with difficulty. Wound closed with drainage.

PATHOLOGY.—In the middle third there was an annular thickening of the appendix wall about ¼ in. long which had caused obstruction of the lumen. Distal to it the appendix was distended and contained foul pus. There was acute inflammation of the mucosa, with areas of gangrene. There was no perforation.

Microscopical examination showed that the submucosa and the muscle coats were infiltrated by a carcinoid tumour in the middle third of the appendix.

PROGRESS.—The patient ran an irregular temperature for a few days, but was discharged quite well on Oct. 5, 1933. He enjoyed good health until November, 1934, when he was admitted to the Newcastle General Hospital with acute osteomyelitis of the mandible, from which he died in spite of operation. At autopsy there was no evidence of any recurrence of the carcinoid tumour.

Case 4.—J. K., male, aged 21 years.

HISTORY.—Admitted on June 10, 1935. Patient complained of gradual onset of aching pain in the umbilical region thirty hours before admission, which soon became very severe and made him roll about and vomit. Had some pain on micturition on the day of admission.

FIG. 218.—Section of part of the tumour from *Case* 4 under low-power magnification, showing the typical masses of carcinoid cells. Some normal glands are seen in the upper right-hand part of the picture. (× 80.)

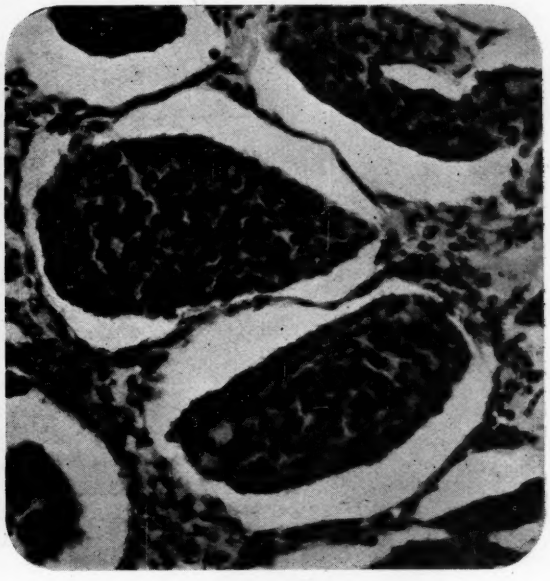

FIG. 219.—Carcinoid cells seen under high-power magnification. (× 250.)

ON EXAMINATION.—General condition fair. Dirty tongue. Temperature 100° F. Pulse 100. Some lower abdominal distension and rigidity. Diffuse tenderness over the lower abdomen, particularly in the right iliac fossa. No mass. A definite tender swelling on the right side was felt per rectum.

OPERATION (June 10, 1935).—Rutherford Morison incision in right iliac fossa. Long appendix hanging over the pelvic brim, distal part swollen. Appendicectomy performed. Wound closed with drainage.

PATHOLOGY.—Appendix about 4½ in. long, terminal 2 in. very distended and inflamed. On opening, it was seen that there was a definite narrow lumen about 2 in. from the tip. At this point there was a marked thickening of all the coats of the appendix right round the lumen for about ½ in. along the length of the appendix. The thickening was partly fibrous, but contained definitely firm yellow nodules of new tissue. Beyond this there were marked inflammatory changes in the appendix, with numerous areas of gangrene of the mucosa. At one point on the anti-mesenteric border near the tip the appendix had almost perforated.

Microscopical examination showed the lumen to be occupied by a typical carcinoid which had diffusely infiltrated the muscle coats (*Figs.* 218, 219).

PROGRESS.—The patient made an uninterrupted recovery. No reply was received to a recent questionaire.

Case 5.—N. W., male, aged 28 years.

HISTORY.—Admitted on Dec. 18, 1936. Patient took ill three days before admission with epigastric pain, which appeared gradually. After a few hours this settled in the right iliac fossa. Vomited several times. Had similar attack four years before, keeping him away from work for ten days.

ON EXAMINATION.—General condition good. Temperature 99° F. Pulse 108. Tongue was dirty and he had a foul breath. Tenderness and rigidity in the right iliac fossa. No mass.

FIG. 220.—*Case* 5. Acute obstructive appendicitis caused by a carcinoid tumour. The carcinoid tumour is seen in the middle third of the appendix obstructing the lumen so that acute inflammatory changes have supervened distal to it.

OPERATION (Dec. 18, 1936).—Rutherford Morison incision in right iliac fossa. Peritoneal cavity opened. Appendix found lying below cæcum and wrapped in omentum. Appendicectomy was performed and the wound closed.

PATHOLOGY.—Appendix about 4 in. long. In the middle third there was an annular thickening of the appendix caused by typical carcinoid tumour (*Fig.* 220). There was obliteration of the lumen, and distal to this the appendix was distended and contained pus. There was acute inflammation of the mucosa, with marked fibrosis of the muscle coats. There was no perforation.

Microscopical examination showed that the thickening was a definite carcinoid which was infiltrating into the muscle coats.

PROGRESS.—The patient made an uninterrupted recovery. Recent letters show him to be in perfect health, with no evidence of any recurrence of the carcinoid.

Case 6.—J. G., male, aged 18 years.

HISTORY.—Admitted on Jan. 20, 1933. Two weeks before admission complained of severe pain in the right iliac fossa which made him vomit. It lasted a day and then gradually got better, and he was able to resume work. Two days before admission pain reappeared but was less severe.

ON EXAMINATION.—General condition fair. Temperature 100° F. Pulse 90. Tender mass palpable low down in the right iliac fossa.

OPERATION (Jan. 21, 1933).—Rutherford Morison incision in right iliac fossa. The mass was found to be thickened cæcum and a coil of small intestine adherent to the parietes. The abdomen was packed off. Dissection showed that there was a very thickened and swollen appendix lying along and adherent to the lateral wall of the cæcum. Appendicectomy was performed, and the wound closed with drainage.

PATHOLOGY.—The appendix was swollen and contained some mucopus. There was an obstruction at the base caused by a definite carcinoid tumour.

Microscopical examination confirmed the opinion that the tumour was a typical carcinoid.

PROGRESS.—The patient ran an irregular temperature after operation for about a week, but eventually made an excellent recovery and was discharged from hospital on Feb. 7, 1933.

He was admitted to the medical wards on May 11, 1933, suffering from acute lymphatic leukæmia, from which he died two days later. A complete autopsy was performed, and no evidence of recurrence of the carcinoid was found.

Case 7.—E. M., female, aged 36 years.

HISTORY.—Admitted to the Eye Department in June, 1927, for cyclitis, and while still an in-patient developed acute appendicitis and was operated upon within twenty-four hours of the onset. Appendicectomy was performed.

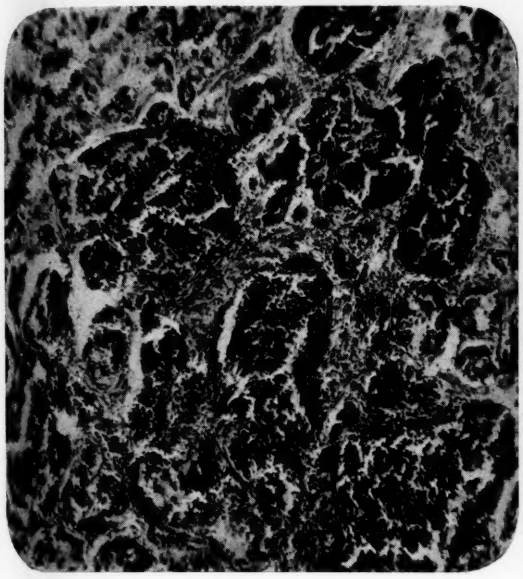

FIG. 221.—Low-power magnification of part of the tumour from *Case* 7. (× 80.)

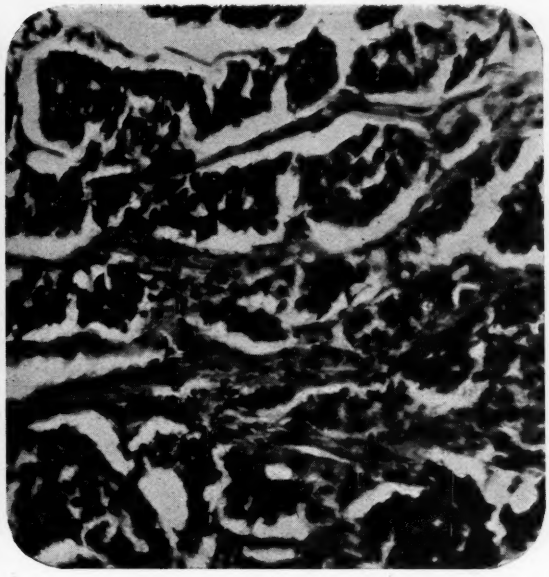

FIG. 222.—High-power view of part of the same tumour, showing the muscular tissue present in the stroma. (× 250.)

PATHOLOGY.—The appendix was the seat of an early catarrhal appendicitis, and at the tip there was a beautiful example of a carcinoid tumour.

Microscopic examination confirmed the macroscopic diagnosis (*Figs.* 221, 222).

PROGRESS.—The patient had an uninterrupted convalescence. No reply received to a recent questionaire.

Case 8.—G. S., male, aged 37 years.

HISTORY.—Admitted on May 17, 1936. Patient complained of pain in the right iliac fossa of two days' duration, and vomiting. No previous attack. Had had a typical duodenal ulcer history for ten years.

ON EXAMINATION.—General condition good. Dirty tongue. Temperature 99·2° F. Pulse 100. Abdomen: tender in right iliac fossa; no mass; no rigidity. Per rectum: nil.

OPERATION (May 19, 1936).—Right paramedian incision. Anterior-wall duodenal ulcer. Also definite acute appendicitis. Appendicectomy. Wound closed.

PATHOLOGY.—Very congested and œdematous appendix. The whole of the mucous membrane was discoloured and softened into a greyish sloughing mass. Peritoneal coat acutely congested. At the tip there was a white firm nodule the size of a pea.

Microscopical examination showed this nodule to be a typical carcinoid. Groups of tumour cells had infiltrated through the muscle and were present in the subserosa.

PROGRESS.—The patient made an uninterrupted recovery. In reply to a recent letter, he states that he is in perfect health.

Case 9.—I. S., female, aged 28 years.

HISTORY.—Admitted on Nov. 4, 1936. Complained of pain in the right iliac fossa, followed by vomiting, twenty-four hours before admission. Gradually getting worse. Had had several previous attacks.

ON EXAMINATION.—General condition fair. Temperature 101° F. Pulse 120. Tender and rigid in right iliac fossa. No mass.

OPERATION (Nov. 4, 1936).—Rutherford Morison incision in right iliac fossa. Appendix adherent to medial side of cæcum. Appendicectomy performed. Wound closed with drainage.

PATHOLOGY.—Appendix was acutely inflamed and thickened. At the tip there was a small fibrous nodule the size of a pea.

Microscopical examination showed the appendix to be acutely inflamed, with ulceration of the mucosa and hæmorrhage and cellular infiltration. These changes were limited to the mucosa and submucosa. At the tip there was a pea-sized carcinoid.

PROGRESS. The patient made an uninterrupted recovery and was discharged on Dec. 12, 1936. Recent inquiry shows her to be in perfect health.

Case 10.—R. G., male, aged 9 years.

HISTORY.—Admitted on Dec. 12, 1936. Had had several attacks of lower abdominal pain during the previous year. Diagnosis: chronic appendicitis.

OPERATION (Dec. 13, 1937).—McBurney incision. Numerous enlarged and calcified glands in the mesentery. Appendicectomy was performed and the wound closed.

PATHOLOGY.—Appendix was macroscopically normal.

Microscopical examination showed that the appendix was normal, but at its tip there was a small carcinoid. There was no evidence of tuberculous infection.

PROGRESS.—There has been no evidence of any recurrence to date.

SUMMARY

1. The literature of carcinoid tumours of the appendix is reviewed.

2. Reports of 10 new cases are given.

3. In 60 per cent of the cases the tumour was responsible for the onset of acute obstructive appendicitis.

My thanks are due to the various members of the Honorary Surgical Staff of the Royal Victoria Infirmary, Newcastle-on-Tyne, for granting me permission to quote cases admitted under their care.

REFERENCES

[1] MERLING, *Jour. de l'Expérience*, 1838.
[2] PRUS, *Thesis of Croizet*, 1865, Paris.
[3] ROKITANSKY, *Med. Jahrb.*, 1867, xiii, 179.
[4] KOLACZEK, *Arch. f. klin. Chir.*, 1875, xviii, 366.
[5] LEICHTENSTERN, *Ziemssen's Handbuch*, 1876, vii, 2, 524.
[6] BIERHOFF, *Deut. Arch. f. klin. Med.*, 1880, xxvii.
[7] BEGER, *Berl. klin. Woch.*, 1882, xix, 616.
[8] ELTING, A. W., *Ann. of Surg.*, 1903, xxxvii, 549.
[9] MOSCHCOWITZ, A. V., *Ibid.*, 891.
[10] ROLLESTON, H. D., and JONES, L., *Amer. Jour. Med. Sci.*, 1906, cxxxi, 951.
[11] LUBARSCH, O., *Virchows Arch.*, 1888, cxi, 280.
[12] BUNTING, C. H., *Bull. Johns Hopkins Hosp.*, 1904, xv, 389.
[13] OBERNDORFER, S., *Frankf. Zeits. f. Pathol.*, 1907, i, 426; *Handbuch der speziellen pathologischen Anatomie und Histologie*, 1929, iv, 717. Berlin: Julius Springer.
[14] GRAHAM, J. M., *Edin. Med. Jour.*, 1913, N.S., x, 30.
[15] WILKIE, D. P. D., *Brit. Jour. Surg.*, 1920–21, viii, 392.
[16] ILLINGWORTH, C. F. W., and DICK, B. M., *A Textbook of Surgical Pathology*, 1935. London: Churchill.
[17] REIMANN, S. P., *Amer. Jour. Med. Sci.*, 1918, clvi, 190.
[18] SIMON, *Ergebn d. Chir. u. Orthop.*, 1916, ix, 291.
[19] MACCARTEY, W. C., and MCGRATH, B. F., *Ann. of Surg.*, 1914, lix, 675.
[20] RAIFORD, T. S., *Amer. Jour. Cancer*, 1933, xviii, 803.

[21] COOKE, H. H., *Arch. of Surg.*, 1931, xxii, 568.
[22] STEWART, M. J., and TAYLOR, A. L., *Jour. Pathol. and Bacteriol.*, 1926, xxix, 136.
[23] WYATT, T. E., *Ann. of Surg.*, 1938, cvii, 260.
[24] SHAW, E. H., *Brit. Jour. Surg.*, 1925–26, xiii, 130.
[25] BURCKHARDT, J. L., *Frankf. Zeits. f. Pathol.*, 1909, iii, 593.
[26] KROMPECHER, E., *Ziegler's Beitr.*, 1919, lxv, 79.
[27] TRAPPE, M., *Frankf. Zeits. f. Pathol.*, 1907, i, 119.
[28] TOENNIESSON, E., *Zeits. f. Krebsforsch.*, 1910, viii, 355.
[29] SALTYKOW, S., *Beitr. z. pathol. Anat. u. allg. Pathol.*, 1912, liv, 559.
[30] HUEBSCHMANN, P., *Rev. méd. de la Suisse, rom.*, 1910, xxx, 317.
[31] SCHIDMT, J. E., *Arch. f. mikr. Anat.*, 1905, lxvi, 12.
[32] GOSSET, A., and MASSON, P., *Presse méd.*, 1914, xxv, 237.
[33] HASEWAGA, T., *Virchows Arch.*, 1923, ccxliv, 20.
[34] DANISCH, F., *Ziegler's Beitr.*, 1924, lxxii, 687.
[35] FORBUS, W. D., *Bull. Johns Hopkins Hosp.*, 1925, xxxvii, 130.
[36] MASSON, P., *Amer. Jour. Pathol.*, 1928, iv, 181; *Ibid.*, 1930, vi, 217.
[37] KULTSCHITZKY, H., *Arch. f. mikr. Anat.*, 1897, xlix, 7.

TWO CASES OF ENDOTHELIOMA OF THE PLEURA

By N. R. BARRETT and J. St. C. ELKINGTON, London

There is no general agreement, amongst authorities upon the subject, that 'endothelioma of the pleura' exists as a pathological or a clinical entity. The majority of those who record cases seem anxious to prove that the condition is really one of primary carcinoma of the lung or bronchus which has spread into the parietal and visceral pleuræ, and in those cases where no primary can be found in the lung it is assumed that the growth is small and has been overlooked. It is not the purpose of this paper to enter into this controversy, but rather to point out and explain some physical signs which we have found in two cases of diffuse new growth of the pleura. These observations may enable the diagnosis to be made sooner, and so save patients a great deal of treatment which is both useless and tiresome.

In both the cases described a careful post-mortem examination was made, and the pathologist's reports are quoted. In neither was a primary tumour demonstrable in the bronchial tree, nor was there any evidence of lymphatic invasion, in spite of the fact that the growths were very extensive. In the second case malignant cells were found in the sputum before death, and these were diagnosed by Professor Dudgeon as from an endothelioma. At the post-mortem examination of this case it was obvious that the malignant tissue was invading the bronchi from without, and paraffin sections showed the mucosa intact in places where collections of malignant cells had penetrated between the cartilaginous rings and extended along the submucosa. The fact that malignant cells were ultimately demonstrable in the sputum is not necessarily an argument in favour of the suggestion that this was a primary bronchial growth.

From the clinical point of view, both these tumours gave physical signs which were difficult to interpret in the early stages.

CASE REPORTS

Case 1.—A woman aged 49.

History.—There was nothing of interest in her past history, and her first symptom had been a mild aching pain in the lateral part of the chest on the left side, which began in August, 1936. Her doctor diagnosed 'pleurisy' and sent her to another hospital, where this opinion was confirmed. A radiograph was taken at this time, and showed an increased opacity in the region where the pain was situated. It was also noted that the left side of the chest moved poorly on respiration and that there was some impairment of percussion note and air entry in the left axilla. During her stay in hospital the pain became worse and more continuous. After resting for six weeks she was discharged and returned to the care of her doctor, who treated her symptomatically. The symptoms, however, got worse, and she sought the advice of another medical man who diagnosed 'spinal trouble' and prescribed massage, exercises, and a belt. This treatment aggravated the condition, and she began to suffer from dyspnœa on exertion, as well as constant pain.

Four months before admission to St. Thomas's Hospital she complained of numbness of the affected side of the chest and the abdomen; the numbness started below the left nipple and gradually spread down to the abdominal wall. She lost weight and suffered

from insomnia due to pain, but there was neither cough nor hæmoptysis. She therefore consulted another doctor, who noted paresis of the intercostal muscles as well as the signs mentioned above and suspected a spinal tumour.

ON ADMISSION (March, 1937).—She was thin and wasted, but otherwise her general condition was still fairly good. The pain and dyspnœa were constant. The left side of the chest moved badly on respiration and the percussion note was impaired in the left upper zone both in front and behind, whilst the whole of the left base was dull. The breath-sounds were distant and in places tubular. There was some scoliosis to the left. The right chest was normal.

The cardiovascular system was normal, and in particular there was no displacement of the apex beat. Nothing abnormal could be palpated in the abdomen, but when the patient was asked to tighten up her abdominal muscles there was an obvious weakness of the whole of the left side of the abdominal wall.

In the central nervous system the motor, reflex, and sensory functions of the cranial nerves and all four limbs were normal. There was wasting and complete paralysis of the intercostal muscles on the left side from the 6th to the 11th inclusive. There was great weakness of the upper segments of the left rectus abdominis and the obliques, but the erector spinæ muscles were acting normally and equally on the two sides. The abdominal reflexes could not be obtained on either side, and there was marked impairment of all forms of cutaneous sensibility on the left side of the trunk in the area supplied by the 6th to the 10th spinal segments. Posteriorly, however, this area of sensory loss did not extend to the midline, but stopped abruptly at the outer edge of the erector spinæ. There were no other abnormal physical signs.

Repeated radiographs were taken and showed an increased opacity in the outer part of the left chest, a general opacity at the left base, and a paralysis of the left side of the diaphragm.

The cerebrospinal fluid was under normal pressure, and there was no evidence of a spinal block. It contained no abnormal constituents and the Wassermann reaction of the blood and fluid was negative.

PROGRESS.—Her condition got steadily worse, she became comatose, and died two months after admission. The paralysis of the intercostal and upper abdominal muscles remained complete, and the sensory loss gradually progressed to total anæsthesia. In spite of this her pain persisted to the end.

At no time during her illness did she suffer from cough or the expectoration of sputum.

POST-MORTEM EXAMINATION.—It was found that the pleural cavity on the left side was totally obliterated by a tough mass of growth having the consistency and appearance of white fibrous tissue. It completely encased the lung and was especially thick at the base, where the diaphragm was also involved. The growth had invaded the pericardium and extended round the great vessels and the hilum of the lung. The bronchi were not involved, and there was no evidence of lymphatic metastases. Outside the chest the organs were normal except that there were metastases in the suprarenals. The paraffin sections taken from various parts of the tumour were diagnosed as fibrosarcoma.

Case 2.—The patient was a seaman aged 49.

HISTORY.—Thirteen months before admission he also had noticed a dull ache in the left chest. It did not amount to pain and was situated in the front of the chest above the nipple and in the axilla. He went to his doctor, who prescribed symptomatic treatment, and at the end of three weeks he was well again. Five months later a pain appeared in the same position and this was followed immediately by an attack of 'influenza', which forced him to stay in bed for a month. The pain was described as a tight feeling which was aggravated by exercise and relieved by rest. His doctor diagnosed a pleural effusion at this time, and although the symptoms did not subside, the patient eventually returned to work on his vessel. It was not long before he had to give up work again because, although he felt well in himself, the 'stitch' in his side was too great a handicap. A month later he began to lose weight, and he was admitted to the Royal Naval Hospital at Chatham for investigation. He remained there for five weeks, during which time he started a dry and non-productive cough. He was unable to sleep because of the pain, which was now present both night and day. This pain was partially relieved by the aspiration of about half a pint of serous fluid from the left pleural cavity. A radiograph taken at this time was reported

as follows: "The right lung is normal. The lower half of the left lung is obscured by an effusion, and the appearances suggest collapse of the lower lobe, possibly due to a bronchial carcinoma." He was transferred to St. Thomas's Hospital.

ON ADMISSION.—His general condition was good. He was not thin or wasted and seemed quite comfortable walking about the ward. Shortly after admission he got a 'cold'

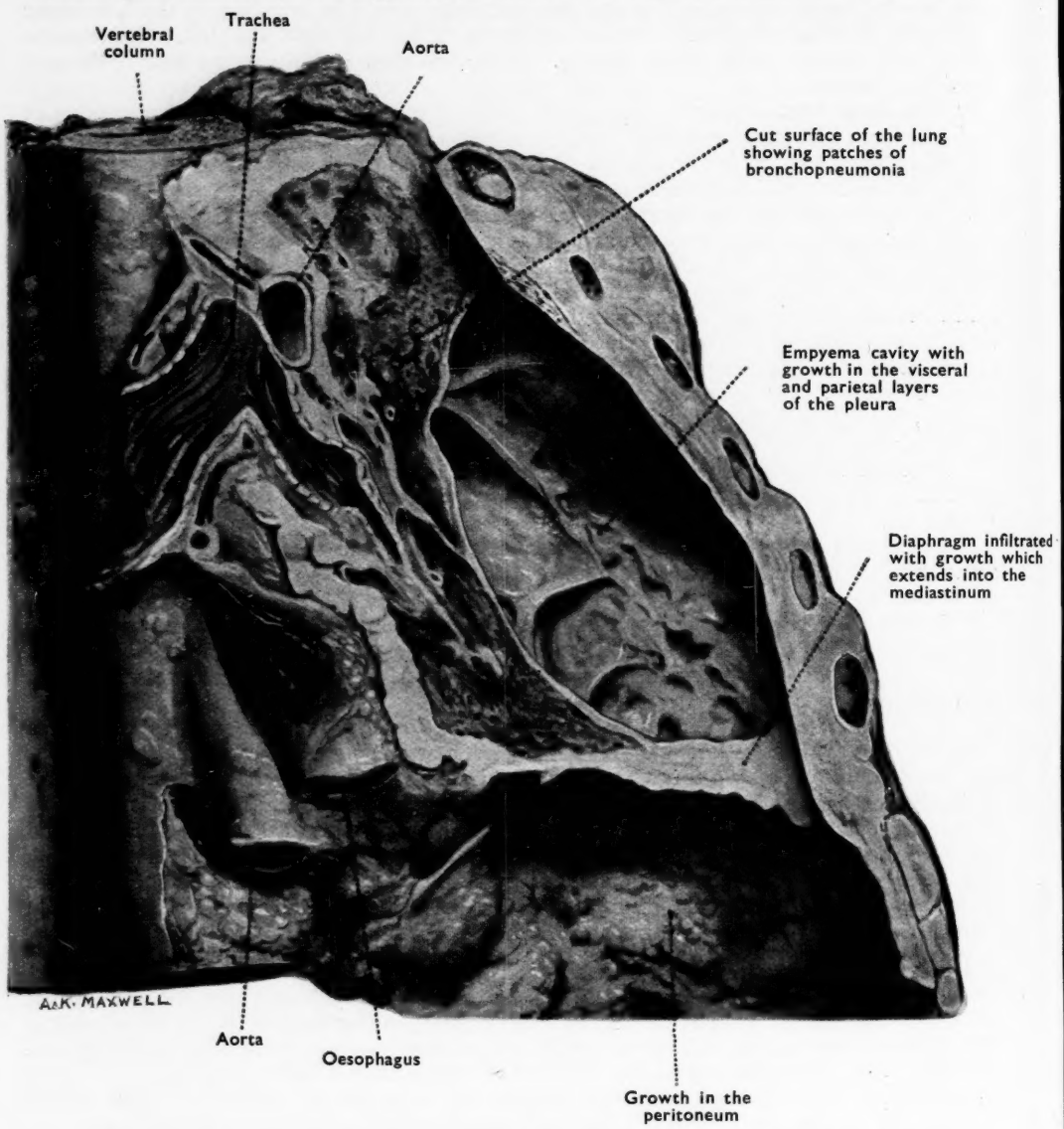
Vertebral column
Trachea
Aorta
Cut surface of the lung showing patches of bronchopneumonia
Empyema cavity with growth in the visceral and parietal layers of the pleura
Diaphragm infiltrated with growth which extends into the mediastinum
A&K. MAXWELL
Aorta
Oesophagus
Growth in the peritoneum

FIG. 223.—*Case* 2. Post-mortem appearance of the posterior parts of the left half of the chest and upper abdominal cavity.

and began to bring up several ounces of purulent sputum every day: this productive cough was present from then until he died.

The right chest was normal, but the left side hardly moved at all on respiration. The intercostal spaces gave an impression of fullness which was absent on the right, and there was a definite but diffuse mass palpable beneath the upper part of the pectoralis major in front. The physical signs were those of a thickened pleura with an effusion at the left base

and a scoliosis towards the left. The fingers were clubbed. The cardiovascular system was normal. In the abdomen the liver edge was palpable an inch below the costal margin and there was a paresis of the muscles of the left abdominal wall.

The signs in the central nervous system were almost identical with those of the first case, except that the intercostal paralysis extended from the second to the eleventh inclusive. There was a similar anæsthetic area involving the left half of the trunk, which extended to the midline in front and ended sharply at the outer edge of the erector spinæ behind. The upper limit of the anæsthesia was in the second intercostal space, and the lower an inch above the pubis. The diaphragm was inactive on the affected side.

There were no other abnormal physical signs. The Wassermann and Kahn reactions in the blood were negative.

Soon after admission a pint of slightly blood-stained fluid was aspirated from the back of the left pleura, and this again produced a temporary diminution of pain and cough. It contained blood and polymorphs, but was sterile on culture.

Radiographs of the chest showed an effusion at the left base and a generalized increase in density of the whole of the lung field.

A bronchoscope was passed, but nothing abnormal was seen. The sputum was examined repeatedly for malignant cells: at first none were found, but towards the end they were present in several specimens and were diagnosed as from an endothelioma.

PROGRESS.—The patient began to go downhill almost immediately; he became cyanosed and dyspnœic, even when lying at rest in bed. The mass in the upper part of the chest increased in size and was easily palpable. He complained that the whole of his chest was painful and tight, and breathing was very difficult. Towards the end large doses of morphia were given for the pain, and he eventually became comatose and intensely cyanosed. A week before he died there was a pericardial rub and pulsus paradoxus, and multiple nodules appeared in the skin. He died three months after admission.

POST-MORTEM EXAMINATION.—The skin of the neck and the upper part of the chest showed a number of small deposits of growth. An enormous mass occupied the whole of the left side of the chest and the mediastinum and passed over towards the right. The heart was almost buried in the mass owing to the extensive involvement of the pericardium, and the left lung and parietal pleura were involved as one, the pleural space being obliterated except in one small area at the back, where an encysted empyema was found. (*Fig.* 223.)

Nodules of growth were visible in several of the intercostal spaces, and particularly in the upper part of the chest anteriorly. Extension of the growth downwards had involved the whole of the left dome of the diaphragm, and the spleen was surrounded by a layer of growth which bound it to the chest wall and the diaphragm. Both kidneys, both suprarenals, and the liver contained metastases. The growth was firm and in some areas even hard. It was of a pale pinkish-buff colour.

Dissection of the left lung showed the growth invading the lung tissue and there was infiltration of the bronchial mucosa in many places, although no part of the bronchial tree exhibited any appearance suggestive of a primary tumour. Apart from the tumour there were extensive patches of bronchopneumonia in the left lung.

There were no secondaries in glands or in the brain.

Histologically the tumour might best be described as a fibrosarcomatous type of endothelioma; that is to say, that whilst for the most part the cells were typical of a fibrosarcoma, yet in those places where they were invading the lung tissue, including the bronchial walls, there was a more sphenoidal type of cell such as is associated with malignant endothelioma. There was no evidence histologically that the tumour was a primary bronchial carcinoma.

COMMENT

The outstanding clinical feature in these two cases was the concurrence of signs pointing to involvement of the respiratory and of the nervous systems.

In each case the respiratory signs pointed to a very marked, progressive, and diffuse thickening of the pleura, with little evidence of involvement of the lung itself.

The nervous signs were strikingly similar in the two cases. They consisted of wasting and complete paralysis of certain of the intercostal muscles on one side,

and also of the upper segment of the abdominal recti and obliques. In each case the diaphragm on that side was immobile.

The sensory disturbance was conspicuous. Its limits were precise, and extended to the midline in front and corresponded accurately with the motor loss

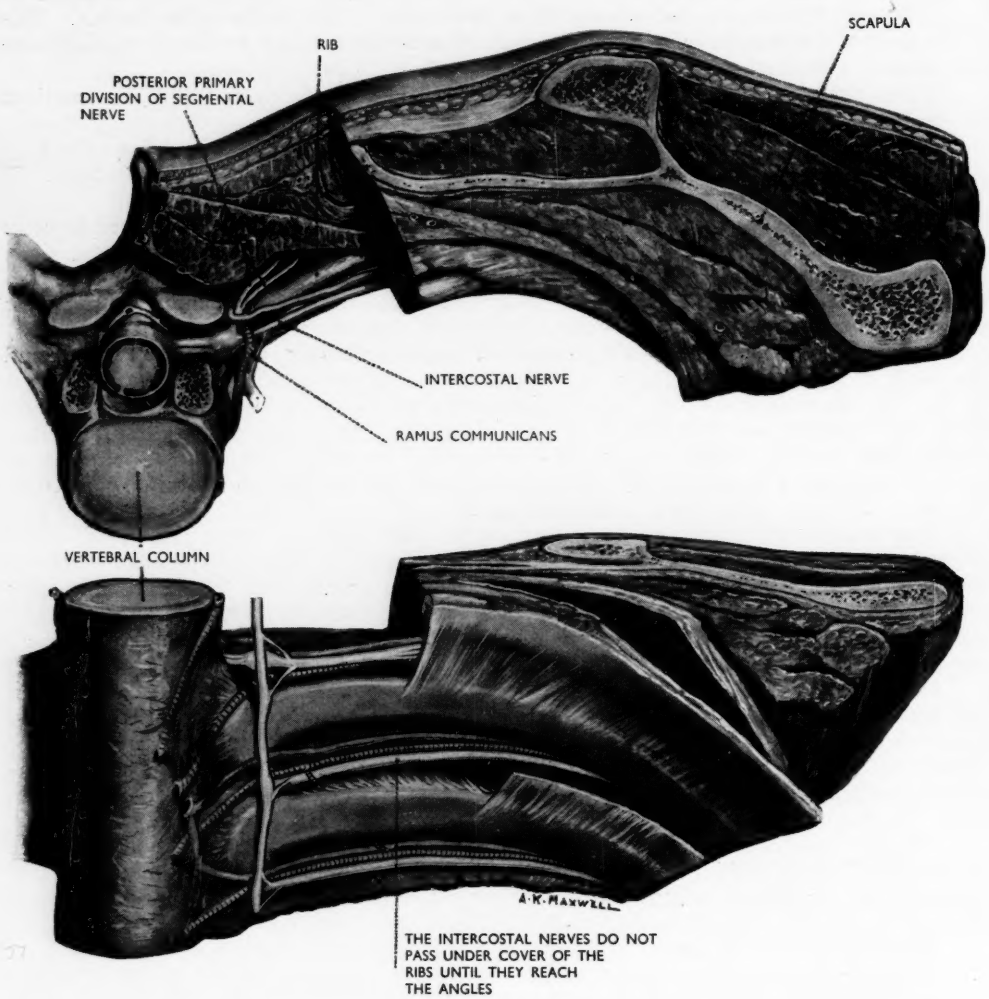

FIG. 224.—Diagrams to illustrate the relationship of the intercostal nerves and the posterior primary divisions of the segmental nerves to the pleura and ribs. (*Drawings made from a dissection in the Anatomical Department of St. Thomas's Hospital.*)

above and below. Posteriorly the sensory loss extended only to the outer edge of the erector spinæ. The sensory loss was severe and equal for touch, pain, and temperature.

In each case the functions of the cranial nerves and sphincters, and the motor, reflex, and sensory functions of both upper and lower extremities, were absolutely normal.

It was clear that the nervous signs could not be explained by a lesion within the spinal canal. The severity of the motor paralysis, and the absolute and clearly

demarcated sensory disturbance involving all modes of sensation equally, were both strong evidence that one was dealing with a root or peripheral nerve lesion. It was inconceivable that both anterior and posterior roots could be thus involved over many segments by disease within the spinal canal without some associated interference with the conducting tracts of the cord.

The escape of the erector spinæ muscles and of the area of skin supplied by the posterior primary divisions of the spinal nerve-roots made it probable that the involvement of the segmental nerves was taking place at a point lateral to the origin of the posterior primary divisions.

The only way in which the intercostal nerves could thus be destroyed seriatim appeared to be by a diffuse new growth involving almost the whole of the inner surface of the thorax on that side.

The dissection shown in *Fig.* 224 was made to demonstrate that the posterior primary division leaves the segmental nerve in such a position that it would be practically impossible for it to be involved by a pathological process in the pleura. But while the posterior primary divisions pass backwards between the neck of the corresponding rib and the articular process of the adjacent vertebra without coming into contact with the pleura, the intercostal nerves pass laterally in immediate apposition to the parietal pleura. The latter are thus exposed either to pressure or invasion by a tumour within the chest.

With regard to the pain complained of by these patients, there were certain features making it unlike the pain commonly associated with disease of the lung or pleura. It was constant and gnawing in quality, unaffected by respiration or coughing; it was situated first in the axilla, and was felt to be in the chest wall itself. It gradually spread to a wide field and was felt in an area which was anæsthetic to external stimuli. All these features suggested that the pain was due to involvement of nerves rather than to irritation of the pleura.

The progress of the disease in each case was marked by increasing cyanosis, and although at first the general health suffered but little, the ultimate deterioration was very rapid. In each case death was preceded by a period of coma lasting several days.

We wish to acknowledge our gratitude to Dr. J. O. Oliver and Dr. D. C. L. Derry, who carried out the post-mortem examinations, and to Professor A. B. Appleton for the dissection shown in *Fig.* 224.

AN OPERATION FOR RECURRENT DISLOCATION (SUBLUXATION) OF THE STERNOCLAVICULAR JOINT

By A. S. BLUNDELL BANKART, London

During the last five years 4 cases of recurrent dislocation or subluxation of the sternoclavicular joint have come under my care. All these cases occurred in young women, who complained of pain and inability to play games, such as tennis, or to indulge in swimming. The dislocation in every case was forwards, and it could be demonstrated by pressing the shoulder backwards. In one of the cases both sternoclavicular joints had dislocated, but only one of them was sufficiently troublesome to require treatment. I could find no account of the treatment of this condition in surgical literature. The following operation was performed in each case.

TECHNIQUE

1. Make a small incision in the lower part of the thigh and remove a strip of fascia lata about 8 in. long and $\frac{1}{3}$ in. wide with a fasciotome. Close the wound and apply dressings.

2. Make an incision 4 or 5 in. long over the inner end of the clavicle, the sternoclavicular joint, and the adjacent part of the sternum, with its centre over the sternoclavicular joint. Reflect the skin flaps upwards and downwards so as to expose fully the front of the joint.

3. With a 1-in. chisel raise the periosteum from the front of the sternum, starting about half an inch away from the joint and penetrating beneath the capsule into the joint.

4. In the same way raise the periosteum and capsule from the inner end of the clavicle.

5. Incise the upper border of the capsule and turn it down with its periosteal attachments.

6. With a chisel separate the insertion of the sternomastoid muscle from the upper border of the sternum and inner end of the clavicle, and penetrate by blunt dissection behind the joint.

7. Thrust a broad flat spatula down behind the joint, and have it held there by an assistant. A malleable brain retractor answers the purpose very well.

8. With a $\frac{1}{8}$-in. twist drill make two holes from before backwards through the inner end of the clavicle. They should be about $\frac{1}{3}$ in. apart and the same distance from the articular margin.

9. Drill two corresponding holes through the sternum on the opposite side of the joint.

10. Pass a loop of silkworm gut through the lower of the two holes in the clavicle, and pull it out from behind the bone with a pair of curved forceps. Thread one end of the fascial strip into the loop, and pull it through to the front of the clavicle (*Fig.* 225).

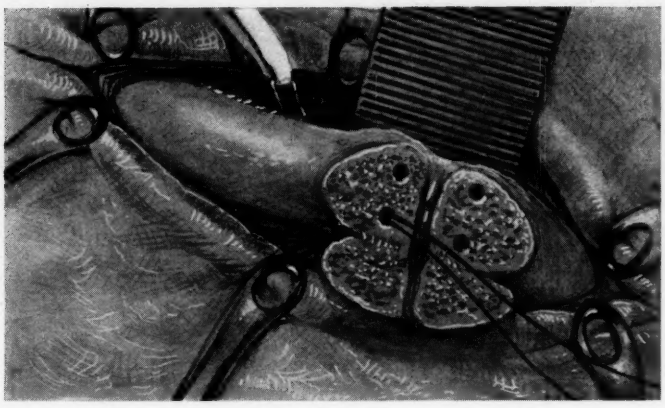

FIG. 225.—Showing the loop of silkworm gut passing through the lower hole in the clavicle. One end of the fascial strip is threaded into the loop, in readiness for pulling through the hole.

FIG. 226.—The other end of the fascial strip being pulled through the upper hole in the clavicle (*see Fig.* 225).

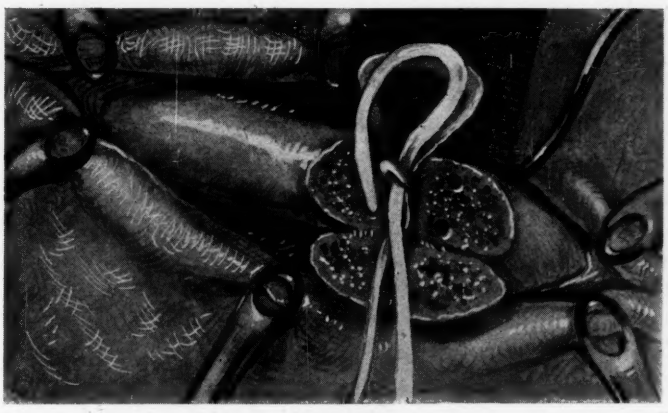

FIG. 227.—Showing the curved aneurysm needle with the two ends of the fascial strip threaded through the eye, prior to being pulled through (*see Fig.* 228).

FIG. 228.—The fascial strip pulled through to the back of the joint.

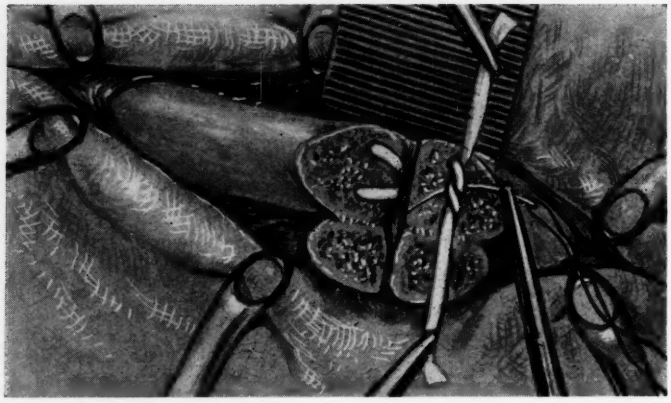

FIG. 229.—The ends of the fascial strip have been brought through the holes in the sternum and tied, and are being held tight by an assistant while the surgeon sews the joint firmly with linen thread sutures.

11. Pass a loop of silkworm gut in the same way through the upper hole in the clavicle, and pull the other end of the fascial strip through to the front (*Fig.* 226).

12. Thrust a blunt instrument into the joint and bore through the capsule at the back of the joint.

13. Pass a large curved aneurysm needle from behind forwards through the joint and, either directly or by means of a silkworm gut loop, pull the two ends of the fascial strip through to the back of the joint (*Figs.* 227, 228).

14. Pass a loop of silkworm gut through the lower hole in the sternum and draw it out from behind the bone with curved forceps. Thread one end of the fascial strip into the loop and pull it through to the front. In the same way pull the other end of the strip through the upper hole in the sternum.

15. Push the inner end of the clavicle firmly backwards. Pull the two ends of the fascial strip tight, and tie them together with the first hitch of a reef knot.

16. Sew the two parts of the strip firmly together with linen thread sutures where they cross over the bone—an assistant meanwhile holding them tight with two pairs of forceps (*Fig.* 229).

17. Lay the free ends of the fascial strip across the front of the joint and sew them to the fascia emerging from the holes in the clavicle. Cut off the excess of the fascial strip.

18. Replace the anterior portion of the capsule which was turned down with its periosteal attachments, and fix it in place with a few catgut stitches.

19. Suture the deep fascia with a continuous suture of fine catgut, and close the skin incision with a subcuticular stitch of salmon gut.

20. Apply dressings and bandage the arm to the side. Remove the subcuticular stitch on the tenth day. Keep the arm bandaged to the side for a month, after which the patient may use the arm as she pleases.

The 4 cases operated on all regained full use of the arm in a few weeks, and in no case has there been any recurrence of the dislocation.

ASPIRATION BIOPSY*

By I. A. B. CATHIE, Manchester

In the present-day treatment of malignant disease the growing use of radiological methods and the realization of the probabilities of tumour radiosensitivity have increased the importance of accurate histological diagnosis. Although in some cases the irradiation is post-surgical, and in these circumstances the pathologist has the operation specimen from which to judge the histology, in others the type of growth must be ascertained from specimens taken by one or other method of biopsy.

In superficial growths the problem is straightforward, and the choice of instrument for obtaining material for section will depend upon the personal preference of the operator and the accessibility of the lesion. In deeper seated tumours, however, the necessity for cutting through healthy tissue, with the possibility of implantation and dissemination of cancer cells, has led to the evolution of different methods of obtaining material for histology. Of these, biopsy by aspiration has perhaps been the most generally adopted, and at the present time the technique, as elaborated by Martin and Ellis[1, 2] and Stewart,[3] is in wide use.

The method is extremely simple, and has the advantage that it is essentially an out-patient procedure, in contrast to the hospitalization necessary for many surgical biopsies from deep-seated tumours. It has also the advantage of rapidity, not more than ten minutes being necessary for obtaining the material, and a further ten minutes for its preparation and examination.

TECHNIQUE

The skin over the swelling is sterilized, and a needle track down to, but not into, the tumour is infiltrated with novocain. An aspirating needle, size 16 standard wire gauge, on a 20-c.c. Record syringe is then introduced through the anæsthetized track into the tumour. While full suction is maintained in the syringe, the needle is pushed once or twice in different planes through the substance of the tumour. Then either the syringe may be removed from the needle, the contents of which are expressed after removal, or suction may be kept up during withdrawal of the needle and aspirated material subsequently removed from the barrel of the syringe.

Material so obtained is smeared between two slides, allowed to dry, and fixed for one minute in absolute alcohol. It is then stained with hæmatoxylin and eosin in the ordinary way, with one minute for each reagent, and mounted in neutral balsam. In this way a rapid and permanent preparation is obtained.

This method of biopsy by aspiration was tried out in a series of cases at the Holt Radium Institute. During a period of nine months 133 swellings were aspirated, from such sites as breast, prostate, and lymph-nodes.

RESULTS

Material aspirated consisted of large numbers of isolated cells. It was found by experience that any definite cytological arrangement was seldom preserved. In

* From The Christie Hospital and Holt Radium Institute, Manchester.

actual diagnosis the number of mitoses was of little significance, and in malignancy the chief diagnostic features were the increase in the average size of the cells and their variation in size and staining in a given microscopic field.

In will be seen from the Table below that a negative report was made 33 times, while pus cells were obtained 9 times.

DETAILS OF THE EXAMINATION OF MATERIAL FROM 133 ASPIRATION BIOPSIES

DIAGNOSIS	SITE												
	Breast	Prostate	Cervical nodes	Parotid	Bone	Thyroid	Axillary nodes	Inguinal nodes	Thigh	Lung	Abdominal wall	Facial mass	Sternal mass
Carcinoma	37	–	–	–	–	1	–	2	2	–	–	–	–
Gelatinous carcinoma ..	2	–	–	–	–	–	–	–	–	–	–	–	–
Pus	2	–	1	1	–	2	–	–	–	–	–	3	–
Chronic mastitis ..	1	–	–	–	–	–	–	–	–	–	–	–	–
Negative	8	5	13	2	–	1	2	2	–	–	–	–	–
Adenocarcinoma ..	–	5	–	5	–	–	–	–	–	–	–	–	–
Scirrhous carcinoma ..	–	5	–	–	–	–	–	–	–	–	–	–	–
Adenoma	–	1	–	–	–	–	–	–	–	–	–	–	–
Squamous carcinoma ..	–	–	9	–	–	–	–	–	–	–	–	2	–
Lymphadenoma ..	–	–	2	–	–	–	1	–	–	–	–	–	–
Oat-cell carcinoma ..	–	–	2	–	–	–	–	–	–	1	–	–	–
Malignant	–	–	4	–	–	–	–	–	–	–	–	–	–
Mixed salivary ..	–	–	1	3	–	–	–	–	–	–	–	–	–
Ptyalin	–	–	–	1	–	–	–	–	–	–	–	–	–
Osteoclastoma ..	–	–	–	–	1	–	–	–	–	–	–	–	1
Papillary carcinoma ..	–	–	–	–	–	–	–	–	–	–	1	–	–
Basal-cell carcinoma ..	–	–	–	–	–	–	–	–	–	–	–	1	–
Totals	50	16	32	12	1	4	3	4	2	1	1	6	1

Thus, of 133 aspirations a diagnosis was made in 91. Of these, 4 were later shown to be incorrect, leaving 87 cases in which the subsequent behaviour of the tumour, or later surgical biopsy, agreed with the aspiration biopsy report.

Little notice was taken of negative or pus reports, but in 5 of the 9 pus cases the condition was shown to be one of acute inflammation, thus increasing the number of

correct diagnoses from 87 to 92, or 69 per cent. This figure does not mean that the remainder were incorrect, as in many cases diagnostic material was not obtained.

Of the 4 wrong diagnoses, one diagnosed as chronic mastitis proved to be a carcinoma, and obviously representative material had not been obtained. What was thought to be a prostatic carcinoma was shown later to be of rectal origin. The bronchial carcinoma was actually a lymphosarcoma. The fourth wrong diagnosis was from a large sternal swelling, which was aspirated three times. The mixed cytology and the presence of giant cells suggested either tuberculosis or an osteoclastoma, but surgical biopsy eventually proved the growth to be a thymoma.

The 33 cases in which a negative diagnosis was made were due either to unsuccessful aspiration or to the nervousness of the patient. Sclerosing growths and

FIG. 230.—Photomicrograph of material from the anterior abdominal wall, showing a secondary papillary carcinoma of the kidney. (× 100.)

FIG. 231.—Photomicrograph of a breast aspiration, showing cancer cells, with polymorphs for comparison of size. (× 120.)

lymphadenoma frequently contain so much fibrous tissue as to render aspiration almost impossible, while in small mobile nodules of growth the difficulty is to know when the needle is in the tumour.

Usually little more can be done in diagnosis than to decide upon malignancy and to indicate the probabilities of the epithelial or sarcomatous nature of the growth. Sometimes, as in four of the cervical nodes, no more could be said than that the tumour was obviously malignant. On rarer occasions, however, greater precision could be attempted. The material from the anterior abdominal wall (*Fig.* 230) was reported as a papillary carcinoma, and it was suggested that the kidney might contain the primary growth. The suggestion proved to be correct. In one of the neck cases the finding of pseudo-cartilaginous material led to the diagnosis of aberrant mixed salivary tumour, and this also was shown to be correct. *Fig.* 231 shows a typical medullary carcinoma of the breast.

In an infant sent to the hospital as a case of angioma, aspiration of clear, cell-free fluid with a high ptyalin content led to the diagnosis of a blockage of Stenson's duct. From the two cases of gelatinous carcinoma of the breast the aspirated material was thick glairy fluid containing a few distorted cells and giving a strongly positive mucicarmine reaction. In some parotids and prostates cells were obtained in a sufficient state of orderliness to permit of the diagnosis of adenocarcinoma.

In this series of cases no untoward incidents occurred during the aspiration. The worst that could be said is that twice while aspirating small neck nodules a large vein was entered. Immediate withdrawal of the needle and pressure on the vein controlled the bleeding easily.

A diagnostic point worthy of note is that patients with cancer experienced very little discomfort from the aspiration, whereas those with benign lesions usually suffered a fair amount of pain.

COMMENT

The figure of 60 per cent correct diagnoses from aspiration biopsy is not far short of the figures obtained by surgical biopsy from superficial lesions. It would appear, therefore, that the method provides a reasonably accurate adjunct to diagnosis, and is worthy of trial when dealing with tumours of doubtful aetiology.

In 1927 Dudgeon and Patrick[4] tried smearing fresh operation specimens on to slides in order to obtain a rapid diagnosis, and they concluded that although difficult until practised, diagnostic preparations were obtainable. In aspiration biopsy the material obtained and difficulties encountered are essentially the same as Dudgeon and Patrick's, while it is not necessary to submit the patient to open operation in order to obtain the material.

The theoretical drawbacks to the procedure are the possibilities of immediate local effects, tumour implantation in the needle track, and cell dissemination by opening up lymph- and blood-vessels. These possibilities are equally applicable to ordinary surgical biopsy.

It is universally agreed that molestation of a melanoma frequently leads to increased growth and metastasis, while scar implants following surgery of breast and bladder cancer are commonly seen. Furthermore, most surgeons can remember a case or cases in which incision of a tumour has led to sudden increase in growth and dissemination. Unfortunately there is usually little proof that metastasis would not have occurred in any case. Kennedy[5] states, with regard to biopsy of the cervix, that it is " the most indefensible violation of any surgical principle which is involved in the treatment of malignancy of the uterus ". He quotes no facts, however.

Always excepting the melanoma, such experimental evidence as exists suggests strongly that incision plays little or no part in metastasis formation. In a series of 637 rats bearing the Flexner-Jobling tumour, Wood[6] found that in the group subjected to biopsies there were metastases in 22·1 per cent at post-mortem, while in the non-biopsied group metastases were present in 21·8 per cent. Thus, there is no significant difference in the formation of metastases in biopsied and non-biopsied tumour-bearing rats, although these results may not be strictly applicable to man. Martin and Stewart,[7] writing of over 3500 aspiration biopsies, were unable to correlate the formation of metastases with the biopsy in any single case. Greenough[8] quotes a series of 72 biopsied breast cancers showing a 54 per cent five-year survival rate.

Of the control group of 310 non-biopsied cases the five-year survival rate was 33 per cent.

With regard to the effects of the local trauma and the implantation of cancer cells in the incision or needle track, the position is simpler. The actual hæmorrhage and severance of tissue continuity are easily controllable. The amount of pain caused by the aspirating needle is relatively slight, and constitutes a valuable diagnostic aid.

Since biopsy in any form is essentially a pre-therapeutic measure, scar implantation is of little importance. If the treatment to be adopted is surgical, then the part to be removed can be made to include the diagnostic incision and any potential nodules of growth in it. If treatment is to be radiological, the incision will be included in the fields of irradiation; and if the treatment cannot inhibit the growth of transplanted cells in the diagnostic incision, it stands little chance of curing the primary growth.

It would thus appear that a good case may be made for biopsy in general, and that where incisional biopsy is for any reason contra-indicated, the diagnostic information to be gained by aspiration biopsy outweighs the theoretical disadvantages of the method.

REFERENCES

[1] MARTIN, H. E., and ELLIS, E. B., *Ann. of Surg.*, 1930, xcii, 169.
[2] MARTIN, H. E., and ELLIS, E. B., *Surg., Gynecol. and Obst.*, 1934, lix, 578.
[3] STEWART, F. W., *Amer. Jour. Pathol.* (Supp.), 1933, ix, 801.
[4] DUDGEON, L. S., and PATRICK, C. V., *Brit. Jour. Surg.*, 1927, xv, 250.
[5] KENNEDY, J. W., *Med. Record*, 1935, cvi, 560.
[6] WOOD, F. C., *Jour. Amer. Med. Assoc.*, 1919, lxxiii, 764.
[7] MARTIN, H. E., and STEWART, F. W., *Amer. Jour. Roentgenol.*, 1936, xxxv, 245.
[8] GREENOUGH, R., quoted by HANFORD and HAAGENSEN, *Ibid.*, 238.

CALCIFICATION OF THE BURSA OF THE CORACOCLAVICULAR LIGAMENT

By H. J. McCURRICH, Hove

Calcified deposits in the region of the shoulder-joint have from time to time been the subject of papers in journals devoted to surgery. Painter[1] described calcification of the subdeltoid bursa, but Codman[2] demonstrated that the calcification usually occurred in the tendon of the supraspinatus muscle deep to the bursa, which was itself unaffected. Brickner[3] described 7 cases, and gave a very full description of the lesion. Carnett[4] stated that " the subacromial bursa is nearly as large as the palm of the hand of the individual in whom it is located, and except for a small projection beneath the deltoid muscle, lies between the acromion process and the head of the humerus, where it is inaccessible for the complete excision so commonly and wrongly advised by numerous writers ". He maintains that these deposits of calcareous matter are in or even under the supraspinatus tendon.

The situation of bursæ in this region is probably not absolutely constant. Bryce[5] in *Quain's Anatomy* states that there is a large bursa superficial to the muscles covering the shoulder-joint, but does not define its limits.

Beesly and Johnston[6] state that " the subacromial bursa lies between the deltoid, the acromion, and the coraco-acromial ligament above, and the capsule of the shoulder-joint and the muscles attached to it below ".

Elmslie[7] describes the bursa as " a tissue space lying in areolar tissue, some strands of which cross the cavity and may divide it into different compartments. It appears, however, to have no definite lining ". He described cases of calcareous deposits in the supraspinatus tendon.

Gwynne and Robb[8] described 10 cases in which calcification had apparently taken place in the supraspinatus tendon in 7 cases, and in the subacromial bursa in 3 cases. Lee,[9] Fabey and Harmon,[10] Tondeur,[11] and others have also described cases.

In the following case, however, it will be observed that the calcified mass occupies a much more mesial position than any deposit described in the above cases. The deposit, which was larger than a hen's egg, appeared to lie between the clavicle above and mesially, and the coracoid process below and laterally.

Hepburn,[12] following a description of the conoid and trapezoid ligaments, adds: " a synovial bursa usually occupies the re-entrant angle between the two ligaments ". And this bursa is referred to in the index as the bursa of the coraco-clavicular ligament. Bryce[13] also refers to this bursa.

It would appear to be probable that in the case to be described, calcification had occurred in this bursa. I have not discovered any description of a similar case. In all the cases described above the calcareous deposit occurs in the region of the head of the humerus, usually in the region of the superior aspect of the great trochanter. In this case, on the other hand, the calcified deposit is in the region of the coracoid process.

From the anatomical descriptions I am not certain whether or not this bursa might be referred to as a prolongation of the subacromial or subdeltoid bursa.

At the time when I operated I had not had an opportunity of consulting the literature on this subject, and so was not aware of the argument as to whether the calcification occurred in the bursa or in the tendon under the bursa. I therefore did not make a careful investigation of this point. No tendon was seen, however, after extracting the calcareous mass. The mass was loosely connected to the coracoid process by areolar tissue containing a few small vessels, and, after shelling it out, a nest of areolar tissue was left immediately beneath the anterior portion of the deltoid muscle, in the depths of which and slightly lateral to its middle the coracoid process could be felt.

CASE REPORT

HISTORY.—H. W. H., a gentleman aged 51, 5 ft. 9½ in. in height, weighing 12 st. 7 lb., and of a generous figure, while playing tennis on a hard court in June, 1936, ran backwards to reach a ball, and in doing so tripped and fell heavily on his left shoulder. He felt some pain and jarring in this shoulder, but continued the game.

He did not seek any medical advice, and had no treatment until October, 1936, when he went to a masseur, who told him he had bursitis and massaged his shoulder.

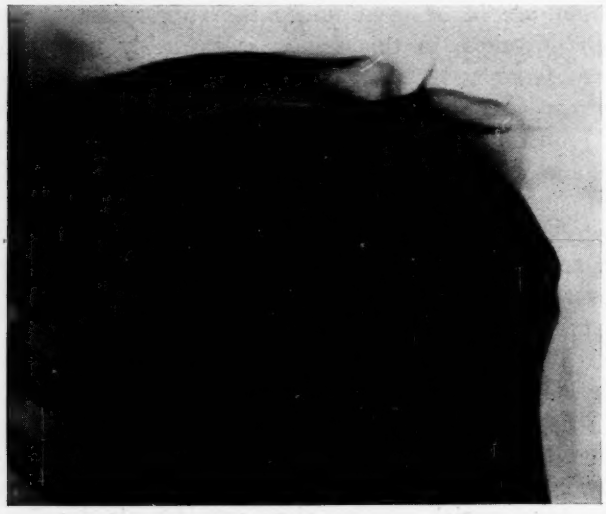

FIG. 232.—Radiograph of the shoulder-joint region, showing the outline of the calcified deposit.

In March, 1937, while visiting Hove, he was seized with acute appendicitis. He consulted Dr. A. H. Greig, who called me in consultation. The patient went to a nursing home immediately, and I removed a gangrenous appendix. He made a smooth recovery from this operation.

He did not mention his shoulder at this time, and neither his doctor nor I was aware that he had injured it.

For three months after this operation he did not have any treatment to his shoulder. He then began to treat his shoulder without medical advice. He applied hot fomentations and other remedies, and had massage. He kept kneading the lump from time to time to remove it.

He thinks that it diminished a little in size until March, 1937 (when he had his appendix removed). After this it increased a little until he restarted treatment, after which it decreased slightly until December, 1937, when he gave up the treatment as useless. For two months after this he did not have pain, but gradually his shoulder became more stiff, and he consulted his doctor in Bournemouth, where he lived.

The disability he complained of was aching of the left arm—the only way to relieve this was to sit slightly bent forwards, with his left arm hanging down as far as possible. He found it difficult to raise his left arm ; he could not get his left hand to his back collar-stud, nor could he use a comb in his left hand.

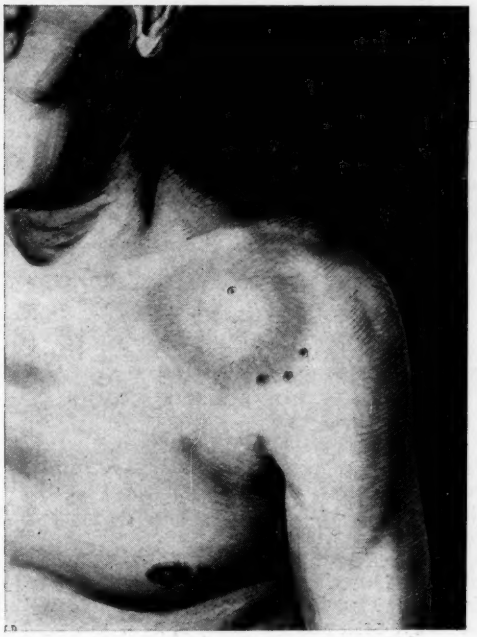

FIG. 233.—Sketch showing the swelling below the outer part of the left clavicle.

He was sent to Dr. Malpas, of Bournemouth, who X-rayed him, and I am greatly indebted to Dr. Malpas for permission to reproduce the radiograph (*Fig.* 232). Dr. Malpas thought that the mass of bone below the clavicle was probably due to an old fracture. He was also

FIG. 234.—The calcified tumour cut through the middle to show its structure.

seen by a surgeon, who told him, so the patient states, that he had an ossifying tumour which might be malignant.

The patient said that he wished to consult me, and I saw him at Hove on March 17, 1938.

ON EXAMINATION.—The superficial appearance of the lesion is well shown in the sketch (*Fig.* 233). There was a globular swelling about 2 in. in diameter below the outer part of the

left clavicle. It was smooth and regular in outline, bony hard, and appeared to be attached to the coracoid process, but not absolutely fixed, a slight degree of movement being possible. The structures superficial to it moved easily over it. The four small circular scars are the result of blisters—he had applied pure acetic acid externally in the hope of dissolving the tumour.

On the history, radiographs, and physical signs, I made a diagnosis of a calcified subacromial bursa, and advised its removal.

OPERATION.—On March 18, the following day, under general anæsthesia I made a 2½-in. incision parallel to the clavicle and over the swelling. The fibres of the deltoid muscle were divided in the direction of their course, and the tumour shelled out without difficulty. It was encapsuled. There was very little bleeding; a few small vessels were ligated, and the wound closed without drainage. Convalescence was uneventful.

PATHOLOGY OF THE TUMOUR.—The accompanying photograph (*Fig.* 234) shows the nature of the tumour. After being sawn in half it was left lying about for a few days before it was photographed, and during this time it shrunk ¼ in., showing that it contained a fair amount of animal matter and moisture.

REFERENCES

[1] PAINTER, *Boston Med. and Surg. Jour.*, 1907, clvi, 345.
[2] CODMAN, *Ibid.*, clix, 533; 1911, clxv, 115.
[3] BRICKNER, *Amer. Jour. Med. Sci.*, 1915, cxlix, 351.
[4] CARNETT, *Radiology*, 1931, xvii, 505.
[5] BRYCE, *Quain's Anatomy*, 11th ed., 1915, iv, 1, 240.
[6] BEESLY and JOHNSTON, *Manual of Surgical Anatomy*, 2nd ed., 13.
[7] ELMSLIE, *Brit. Jour. Surg.*, 1932, xx, 190.
[8] GWYNNE and ROBB, *Austral. and N. Z. Jour. Surg.*, 1934–5, iv, 153.
[9] LEE, *Colorado Med.*, 1936, xxxiii, 316.
[10] FABEY and HARMON, *Amer. Jour. Roentgenol.*, 1937, xxxviii, No. 5, 707.
[11] TONDEUR, *Jour. de Chir. Bruxelles*, 1936, xxxv, 352.
[12] HEPBURN, *Cunningham's Text-book of Anatomy*, 3rd ed., "Articulations", 275.
[13] BRYCE, *Quain's Anatomy*, 11th ed., 1915, iv, 1, 236.

THE NERVE-SUPPLY OF THE GASTRO-ŒSOPHAGEAL JUNCTION

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THE gastro-œsophageal junction receives a supply from both sympathetic and parasympathetic elements of the autonomic nervous system, and, as the arrangement is somewhat complex, it will simplify matters if the nerve-supplies to the lower end of the œsophagus and upper end of the stomach are considered separately. The following observations are based on a series of 14 dissections of infants which were performed with the aid of a dissecting microscope.

THE NERVE-SUPPLY TO THE LOWER END OF THE ŒSOPHAGUS

1. Sympathetic.—The sympathetic supply is derived partly from the thoracic portions of the gangliated sympathetic trunks and partly from the splanchnic nerves, while a few filaments from the sympathetic plexuses around the left gastric and inferior phrenic arteries can be traced upwards on to the lower end of the œsophagus.

In every case the lower œsophagus receives delicate filaments from both sympathetic gangliated trunks (*Figs.* 235–239). These arise from the 6th to the

KEY TO ILLUSTRATIONS

1, Gangliated sympathetic trunk; 2, Greater (thoracic) splanchnic nerve; 3, Lesser (thoracic) splanchnic nerve; 4, Sympathetic nerves (or plexus) around left gastric artery; 5, Sympathetic nerves (or plexus) around right inferior phrenic artery; 5A, Right inferior phrenic nerve described on p. 339 of text; 6, Sympathetic nerves (or plexus) around left inferior phrenic artery; 7, Œsophageal branches derived from the gangliated sympathetic trunk or splanchnic nerves; 8, Para-aortic nerve; 9, Sympathetic fibres to gastro-œsophageal junction which come from above the diaphragm; 10, Filament passing to junction from terminal part of left greater splanchnic nerve; 11, Hepatic plexus; 12, Filaments passing to gastro-œsophageal junction and stomach from hepatic plexus, between layers of lesser omentum; 13, Loop connecting branches of hepatic and left inferior phrenic plexuses around gastro-œsophageal junction; 14, Hepatic branch(es) of anterior vagal trunk; 15, Gastric branches of anterior vagal trunk; 16, Cœliac branch of posterior vagal trunk; 17, Gastric branches of posterior vagal trunk; 18, Posterior pulmonary plexus; 19, Thoracic cardiac nerves; 20, Cœliac plexus; 21, Intermesenteric nerves; 22, Renal plexus; 23, Ovarian nerves; 24, Superior hypogastric plexus (presacral nerve); 25, Hypogastric nerve; 26, Inferior hypogastric plexus (pelvic plexus); 27, Long colonic nerves from inferior hypogastric plexus or hypogastric nerve; 28, Short colonic nerves from inferior hypogastric plexus; 29, Branch(es) to pulmonary plexus from thoracic part of gangliated sympathetic trunk; 30, Direct connection between the right vagus and greater splanchnic nerves; 31, Phrenic nerve; 32, Phrenic ganglion; 33, Lumbar splanchnic nerve(s).

A, Suprarenal gland; B, Bladder (split in median sagittal plane and halves displaced laterally); C, Colon; D, Duodenum; F, Uterine tube; G, Œsophagus; H, Liver; K, Kidney; L, Lung; O, Ovary; P, Pericardium; R, Rectum, S, Stomach; T, Thymus; U, Ureter; W, Uterus; RV, Right vagus; LV, Left vagus.

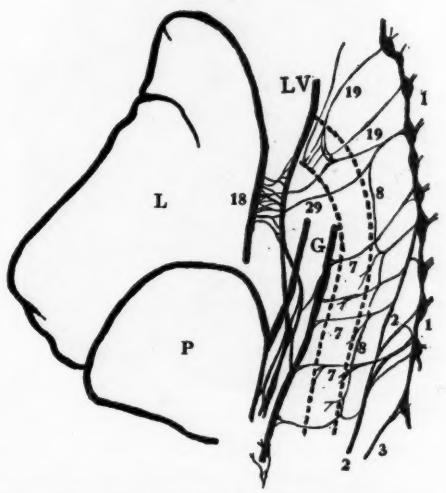

FIG. 235.—A drawing of an actual dissection. The left lung has been drawn over towards the right side. (Lettering and numbering as in key.)

9th or 10th thoracic ganglia, and they often give off aortic branches, but the descending thoracic aorta is more constantly supplied by a regular series of minute branches which are given off from the gangliated trunks and pass alongside the intercostal arteries to the parent vessel. The lower œsophageal filaments are usually more numerous on the left side—in one case examined five were present.

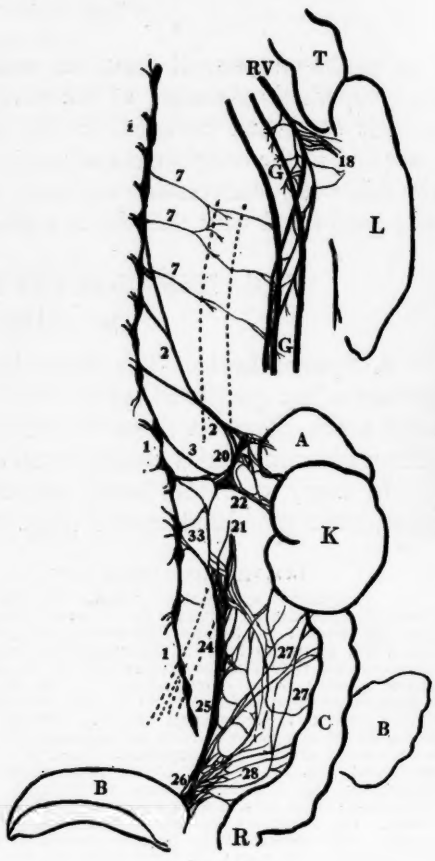

FIG. 237.

FIG. 236.

FIG. 236.—A dissection showing the relationship of the right vagus to the lower œsophagus. The two filaments arising from the gangliated trunk and the filament from the greater splanchnic nerve (seen overlying narrow strip of black paper) supplied both the descending aorta and the œsophagus, but in order to display the right vagus the œsophagus had to be displaced and rotated to the left, which entailed division of the œsophageal filaments.

FIG. 237.—A line drawing of the dissection shown in *Fig.* 236. The upper set of dotted lines represents the descending thoracic aorta; the lower set indicates the line of the iliac arteries.

Lettering and numbering as in key in *Fig.* 235.

On both sides they unite with the vagal plexus, or penetrate the œsophageal wall directly. In four cases (3 left; 1 right) a larger nerve was found arising from the

FIG. 238.

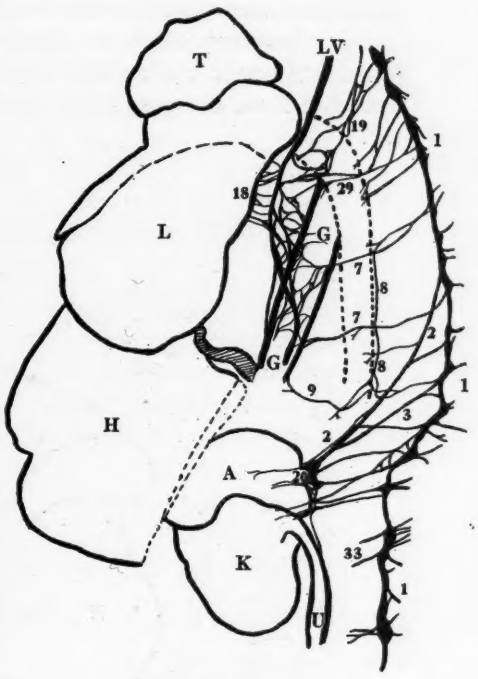

FIG. 239.

FIG. 238.—A dissection showing the left vagus nerve, the left half of the œsophageal plexus, and the thoracic and lumbar parts of the gangliated sympathetic trunk and their branches. In this specimen there were two lower œsophageal sympathetic branches on the left side, one from the gangliated trunk and another from the greater splanchnic nerve; black paper has been placed behind these filaments. They are seen to be connected by a vertical filament which is part of the *para-aortic* nerve described in the text; the upper part was broken in attempting to place black paper behind it. Note that the left vagus nerve inclines *posteriorly* in this specimen and forms the major part of the posterior vagal trunk: normally the reverse is the case, and so far no other record of this interesting anomaly has been traced. To display these structures the thymus, left lung, liver, stomach, left suprarenal, and left kidney have all been displaced to the right, while the left half of the liver has been removed.

FIG. 239.—A line drawing of the dissection shown in *Fig.* 238. The cross-hatched area represents the cut edge of the diaphragm.

Lettering and numbering as in key in *Fig.* 235.

region of the 6th or 7th thoracic ganglia and passing obliquely downwards to enter the œsophageal plexus just above the diaphragm (*Figs.* 238, 239); in each case this nerve supplied additional filaments both to the aorta and to the diaphragm.

Both the greater, and occasionally the lesser, thoracic splanchnic nerves contribute minute branches to the lower œsophagus (*Figs.* 235–237); and on the right side in two cases a larger branch formed a direct connection between the greater splanchnic and vagus nerves at the level of the 7th thoracic vertebra (*Fig.* 240).

In three specimens an interesting arrangement was discovered on the left side (*Figs.* 235, 238, 239). The thoracic cardiac, pulmonary, aortic, and œsophageal branches from the gangliated trunk were united by vertical filaments in practically

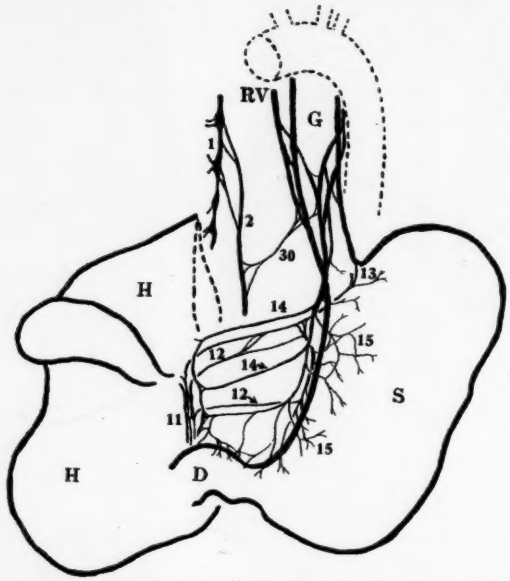

FIG. 240.—A drawing of an actual dissection. The left lobe of the liver has been removed, and the cut edges are indicated by dotted lines. (Lettering and numbering as in key in *Fig.* 235.)

linear series, which together formed a delicate strand lying alongside the postero-lateral aspect of the descending aorta. This *para-aortic* nerve terminated above the diaphragm by supplying filaments to the œsophagus, aorta, and diaphragm.

2. Parasympathetic.—The parasympathetic supply is derived entirely from the vagi. Below the roots of the lungs both vagi usually divide into from two to four main branches, which lie in close relationship to the lower œsophagus. On the right side three are commonly found (*Figs.* 236, 237), and on the left side two (*Figs.* 235, 238), and all are interconnected by finer twigs so that a plexus is formed which surrounds the œsophagus. The right and left groups of vagal fibres pursue an oblique course, so that the former come to lie on the posterior and the latter on the anterior aspect of the viscus,* but one or two small branches from the right vagus join the anterior half and the left vagus contributes a branch or branches to the posterior half of the plexus. This œsophageal plexus is joined by sympathetic filaments, as has already been described. Immediately above the diaphragm, or

* Note that in the dissection shown in *Fig.* 238, the arrangement of the left vagus is abnormal, as it inclines posteriorly instead of anteriorly (see legend under figure).

actually within the œsophageal hiatus, the branches of the plexus reunite into one or two main trunks, which enter the abdomen (*Figs.* 236, 238). These lie anterior and posterior to the gastro-œsophageal junction, and though there is no constant number, it is unusual to find more than two on each aspect, and sometimes there is only one. Each trunk contains a proportion of fibres from both vagi, though right and left fibres preponderate in the posterior and anterior trunks respectively.

Summary.—The sympathetic supply for the lower œsophagus comes from: (*a*) The gangliated trunks between the 6th and 9th or 10th thoracic ganglia; (*b*) The greater, and occasionally the lesser, thoracic splanchnic nerves; (*c*) The para-aortic nerve (when present); and (*d*) The plexuses around the left gastric and inferior phrenic arteries.

The parasympathetic supply is from the vagi.

THE NERVE-SUPPLY TO THE UPPER END OF THE STOMACH

1. Sympathetic.—The sympathetic supply is derived mainly from the cœliac plexus, but it is possible that a small number of sympathetic fibres may come from above through the œsophageal plexus (*Fig.* 241). In a few cases some filaments from the termination of the left greater splanchnic nerve or from the upper end of the left lumbar gangliated trunk are conveyed directly to the gastro-œsophageal junction (*Fig.* 244).

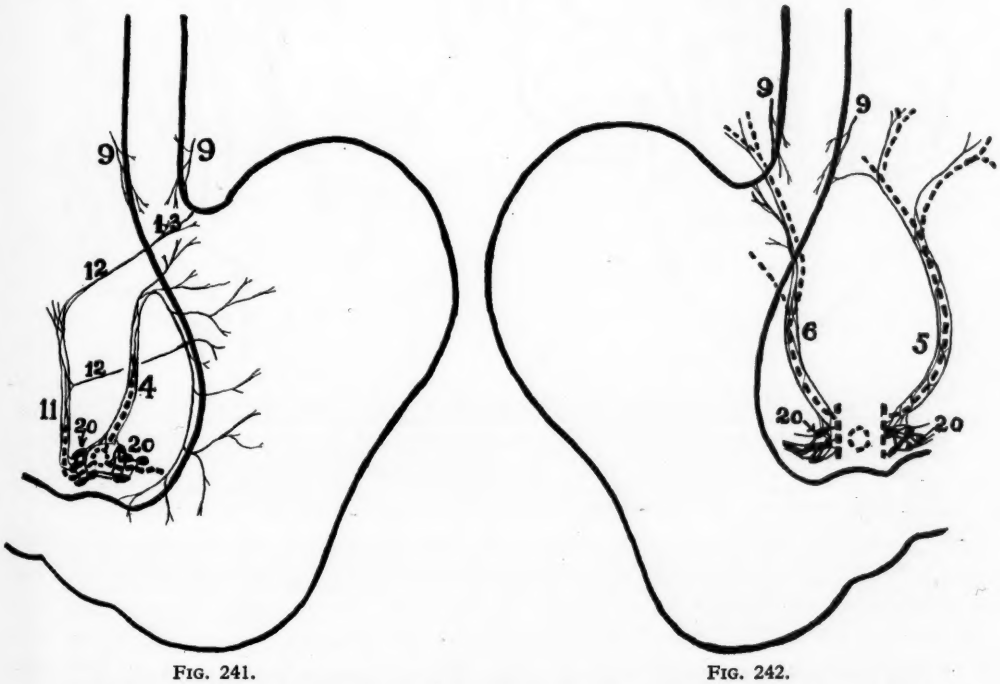

FIG. 241. FIG. 242.

FIG. 241.—A diagram, based on a dissection, representing the sympathetic supply to the gastro-œsophageal junction and stomach from the hepatic and left gastric plexuses. The dotted lines represent the cœliac axis and its branches—hepatic, splenic, and left gastric.

FIG. 242.—A diagram, based on a dissection, showing how sympathetic filaments may reach the gastro-œsophageal junction along the inferior phrenic plexuses. The dotted lines represent the abdominal aorta and its inferior phrenic branches. The dotted circle represents the site of origin of the cœliac axis.

Lettering and numbering as in key in *Fig.* 235.

Most of the supply from the cœliac plexus reaches the cardia alongside the left gastric, inferior phrenic, and hepatic arteries, and the relative importance of these supplies appears to vary from case to case.

The left gastric branches, one to four in number, lie near the artery, and as they approach the cardia they divide into a small proximal group, which supplies the gastro-œsophageal junction, and one or two larger branches, which pass distally along the lesser curvature of the stomach (*Fig.* 241). Another branch often encircles the junction anteriorly, and becomes directly continuous with a branch from the left inferior phrenic plexus (*Figs.* 241, 244, 247, 248).

FIG. 243.

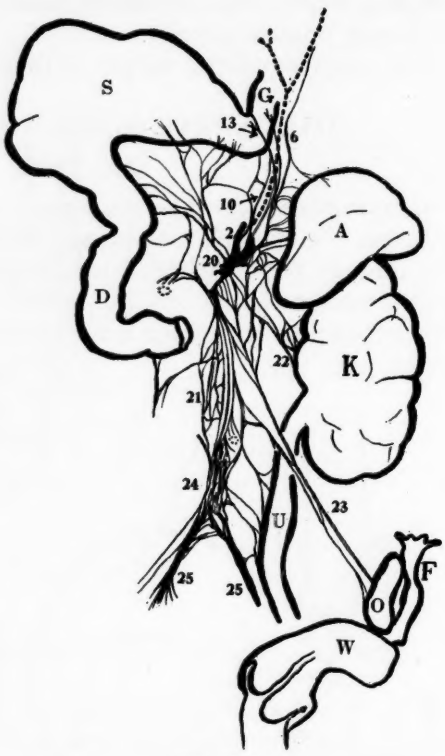

FIG. 244.

FIG. 243.—A drawing of an actual dissection showing a type of right inferior phrenic nerve which has been found in three dissections in this series. It is seen arising from the upper part of the right cœliac ganglion by three small rootlets. At first it lies near the inner side of the corresponding artery (indicated by a dotted line), and then turns to the left, behind the venacaval foramen (indicated by the large dotted circle), to reach the œsophageal hiatus, where it divides into a larger hepatic branch, and smaller filaments which supply the gastro-œsophageal junction: the hepatic branch passes to the liver between the layers of the lesser omentum. The two smaller dotted circles represent the positions of the cœliac axis and superior mesenteric artery.

FIG. 244.—A drawing of an actual dissection showing filaments proceeding to the gastro-œsophageal junction from (*a*) the left inferior phrenic plexus and (*b*) the termination of the left greater splanchnic nerve. The stomach has been drawn over to the right side. The course of the left inferior phrenic artery is indicated by a dotted line.

Lettering and numbering as in key in *Fig.* 235.

The left inferior phrenic plexus gives off one or two branches directly to the cardiac orifice, and one of these usually passes to the right and unites with a twig from the plexus accompanying the left gastric artery, or with a branch to the cardia from the hepatic plexus, forming a loop around the junction of the stomach and œsophagus (*Figs.* 241, 244, 247, 248). The presence of this sympathetic pathway

to the cardia from the left inferior phrenic plexus, which was described as long ago as 1860 by Kollman, has obviously been overlooked, and matters are still further complicated by the fact that other sympathetic pathways exist. Thus of the two or three gastric branches which proceed from the hepatic plexus to the lesser curvature between the two layers of the lesser omentum, the uppermost always runs towards the cardiac orifice, and supplies one or more filaments to it (*Figs.* 241, 247, 248). It is important to realize that this nerve in most of its course is not near the left gastric artery, though it always comes into relationship with its œsophageal branches. Further, in several cases filaments from the upper end of the left lumbar gangliated trunk have been seen to supply the gastro-œsophageal

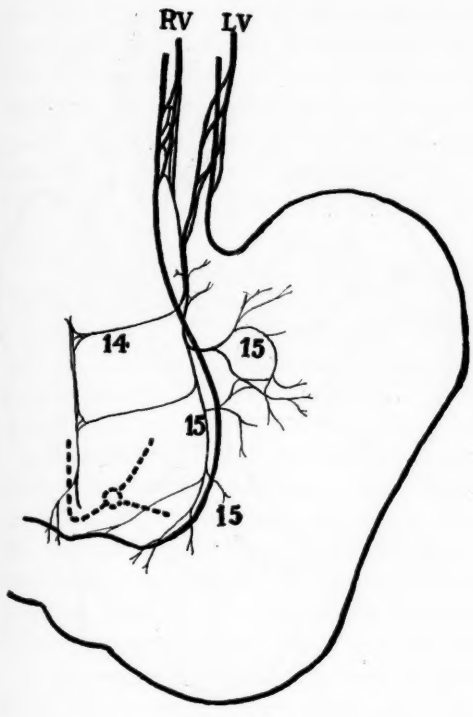

FIG. 245.

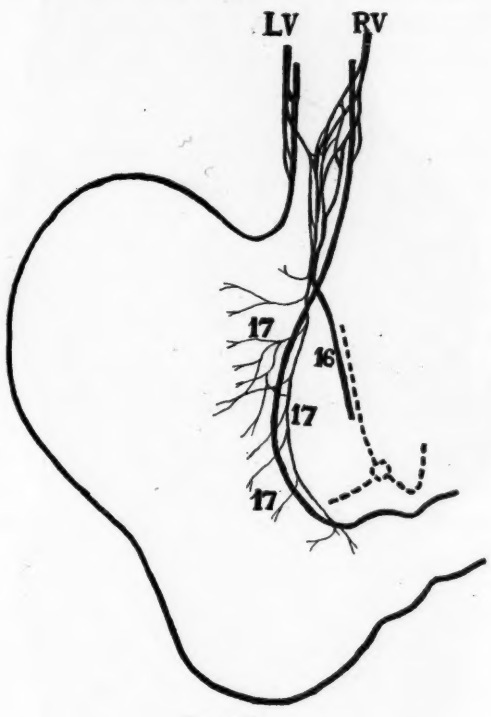

FIG. 246.

FIG. 245.—A diagram, based on a dissection, showing the formation of the anterior vagal trunk and its branches of distribution. The dotted lines represent the cœliac axis and its branches.

FIG. 246.—A diagram, based on a dissection, showing the formation of the posterior vagal trunk and its branches of distribution. The dotted lines represent the cœliac axis and its branches.

Lettering and numbering as in key in *Fig.* 235.

junction, while in two specimens another twig was contributed by the terminal part of the left greater splanchnic nerve; these reach the stomach directly, or unite with the nerves alongside the left inferior phrenic artery (*Fig.* 244). In several cases filaments were supplied to the junction from the right inferior phrenic plexus. There are usually two groups of right inferior phrenic nerves, one consisting of delicate filaments accompanying the artery (*Fig.* 242), and the other, often a single nerve arising from the upper part of the right cœliac ganglion, which lies a little apart from the artery on its inner side (*Fig.* 243). This latter nerve passes towards the venacaval foramen and then turns to the left, behind the foramen, to reach the

œsophageal hiatus in the diaphragm. It supplies one or more filaments to the gastro-œsophageal junction, but the main part of the nerve passes to the liver high up between the layers of the lesser omentum. Before turning to the left behind

FIG. 247.

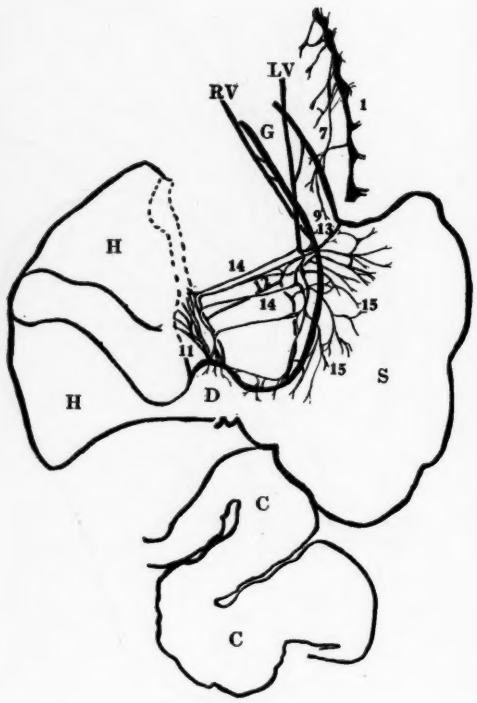

FIG. 248.

FIG. 247.—A dissection showing the anterior vagal trunk and its branches. The six transversely directed branches from the lesser curvature all lay between the layers of the lesser omentum. Three were vagal in origin, and the others were derived from the hepatic sympathetic plexus and passed to the stomach. The left half of the liver has been removed.

FIG. 248.—A line drawing of the dissection shown in *Fig.* 247.

Lettering and numbering as in key in *Fig.* 235.

the venacaval foramen it communicates with a branch of the right phrenic nerve which accompanies the right inferior phrenic artery: on this anastomotic branch

or branches the phrenic ganglion is sometimes situated (*Fig.* 243). Part of the branch of the phrenic nerve accompanying the right inferior phrenic artery ends in the cœliac ganglion.

On both sides the final ramifications of the phrenic nerves and inferior phrenic plexuses appear to communicate, and theoretically this could provide alternative pathways for gastric innervation, but this matter cannot be settled by ordinary dissection.

2. Parasympathetic.—The parasympathetic supply is carried by the vagi. Their mode of entering the abdomen has been described, and now it is necessary to describe as briefly as possible their behaviour in the first part of their abdominal course. The anterior trunk, which may be single or double, divides near the proximal end of the lesser curvature into: (*a*) hepatic, pyloric, and cœliac branches, which run towards the porta hepatis between the layers of the lesser omentum, and then pass upwards or downwards to their destination; and (*b*) a group of gastric branches, some of which radiate to the cardiac orifice and across the fundus and upper part of the body, while one or two larger divisions proceed along the lesser curvature, where their further course does not concern us here (*Figs.* 245, 247, 248).

The posterior vagal trunk (or trunks) also divides into two main sets of branches (*Fig.* 246); the smaller group is distributed to the cardiac orifice and stomach in a manner somewhat similar to the anterior group, but the larger part of the nerve continues downwards and to the right, near to the left gastric artery, and ultimately ends in the cœliac plexus; this is an important relationship.

Summary.—The sympathetic supply to the upper end of the stomach may reach it by several pathways: (*a*) Through the left gastric plexus; (*b*) By way of the left, and occasionally the right, inferior phrenic plexuses; (*c*) Directly from the hepatic plexus by a branch which passes between the layers of the lesser omentum; (*d*) From the upper part of the left lumbar gangliated trunk or the terminal part of the left greater splanchnic nerve; and (*e*) A small number of fibres may come from above the diaphragm through the œsophageal plexus.

The parasympathetic supply is contained in the vagi.

The lower end of the œsophagus and the upper end of the stomach therefore receive nerve-supplies from multiple sources, but this is a characteristic of autonomic innervation, as is well exemplified in the case of the heart, kidney, testis, colon, etc. Whether or not all the pathways described transmit impulses to or from the gastro-œsophageal junction it is impossible to tell by dissection, but there is no doubt that they all supply the junction, or the parts of the alimentary tract immediately adjacent to it.

SURGICAL SIGNIFICANCE

Within recent years the arrangement of the nerve-supply to the gastro-œsophageal junction has assumed practical importance since Knight in 1935 suggested a method of sympathectomy for the relief of achalasia of the cardia and cardiospasm. This procedure—excision of the left gastric artery with its surrounding fatty and nervous tissue—has now been tried on a number of patients, but with the

exception of a few cases (Adamson, Gask and Ross, Learmonth, etc.) the results have not been satisfactory. Gask and Ross state: "The operation has proved to be, on the whole, a disappointment, for out of 8 cases of which we have personal knowledge, only 1 patient has been completely relieved, 1 has derived some benefit, but the others, after a short period of improvement, have all returned to their pre-operative state, and some have continued to deteriorate still further." Assuming that the operation is based on justifiable premises, there must be some reason for this failure to achieve the desired relief in the majority of cases. Gask and Ross suggest that there are two possible explanations for the discrepancy between experimental and operative results. They point out that section of the vagi, as performed by Knight in his experimental animals, does not produce a condition exactly analogous to the pathological state found in true achalasia, which is characterized by degeneration and finally destruction of Auerbach's plexus. Injection of novocain around the stellate ganglion increases peristaltic activity in the normal œsophagus, but produces no effect if Auerbach's plexus is destroyed. It seems, therefore, that once this plexus is degenerated or destroyed, the œsophageal musculature cannot overcome the resistance of a denervated cardiac sphincter when it has recovered its natural tonus. Alternatively they suggest that the sympathetic supply to the sphincter may not always come entirely from below the diaphragm, and in support of this view they quote a successful operation performed by Craig, Moersch, and Vinson, who removed the inferior cervical and first and second thoracic ganglia on both sides in a woman suffering from cardiospasm, which had failed to respond to all the usual methods of treatment. As White points out, however, the nerve-supply to the so-called cardiac sphincter is not derived from the lower cervical and upper thoracic ganglia, and thus the favourable outcome of the operation is difficult to explain on the grounds of interruption of the efferent pathway to the sphincter. He suggests that the benefit secured may have been due to interruption of visceral afferents rather than to paralysis of the cardiac sphincter.

But by amending Gask and Ross's second suggestion to include *all* alternative sources of nerve-supply to the cardiac orifice it is easy to explain, on purely anatomical grounds, why Knight's operation will fail in its object in many cases. In this respect three points require emphasis:—

1. The sympathetic nerve-supply to the gastro-œsophageal junction in man comes from several sources, which differ from case to case in relative importance, and removal of the left gastric artery with its surrounding fatty and nervous tissues will not destroy completely the sympathetic nerve-supply to the junction.

2. The nerve structures in relationship to the left gastric artery are entirely different within a comparatively circumscribed area near the cardia.

3. Widespread removal of the left gastric artery, as suggested and figured by Knight, might produce the opposite effect to that intended—namely, a complete, or almost complete, interruption of the parasympathetic supply, and only a sub-total sympathetic denervation. Thus the patient's condition might be aggravated rather than alleviated.

The exact nerve structures destroyed could vary considerably, depending on the site and extent of the arterial resection (*see* Table on p. 343).

There is no necessity to multiply these possibilities. They do prove, however, that very different results could be produced by relatively slight variations in the site and amount of tissue removed, and they help to explain why certain operations

succeed while others fail. It is possible that in the successful cases the bulk of the sympathetic supply to the cardiac orifice was conveyed by nerves alongside the left gastric artery, or that the œsophageal and gastric branches of the vagi escaped destruction. In unsuccessful cases the more important sympathetic nerve-supply may have been contained in some of the other alternative pathways, or the vagi may have been cut. Where the patient's condition deteriorated after operation it is interesting to speculate whether division of the parasympathetic supply to the cardia may have been a factor of no less importance than continued degeneration of Auerbach's plexus in the further progress of the malady.

NERVES DESTROYED ACCORDING TO THE ARTERIES RESECTED.

I. If the left gastric artery were completely removed, along with its œsophageal branch(es)—

The following nerves would be destroyed :—

1. The sympathetic fibres accompanying the artery.
2. The large division of the posterior vagal trunk which passes alongside the artery to the cœliac plexus.
3. The sympathetic branch which passes from the hepatic plexus, high up between the layers of the lesser omentum, to the cardiac orifice.
4. The hepatic branch of the anterior vagal trunk which lies close to the above nerve.
5. The main anterior and posterior vagal trunks would be in grave danger and would probably be cut, for both incline towards the right after passing through the œsophageal hiatus, and neither is far removed from the termination of the artery and its œsophageal branch(es).

The following nerves would escape :—

1. The branches to the cardiac orifice from the inferior phrenic plexuses.
2. Branches to the cardia from the left lumbar sympathetic gangliated trunk or from the terminal part of the left greater splanchnic nerve.
3. The lower thoracic œsophageal branches from the gangliated sympathetic trunks and splanchnic nerves, including the para-aortic nerve, all of which supply filaments to the termination of the œsophagus.
4. Twigs from the œsophageal vagal plexus which are actually embedded in the muscular coat of the lower œsophagus.

II. If only the middle or proximal part of the left gastric artery was removed—

The following nerves would be destroyed :—

1. The sympathetic fibres accompanying the artery.
2. The large division of the posterior vagal trunk passing alongside the artery to the cœliac plexus.

The following nerves would escape :—

1. The branch to the cardia from the hepatic plexus.
2. The branches from the inferior phrenic plexuses, the left lumbar sympathetic trunk, and the left greater splanchnic nerve.
3. The main vagal trunks.
4. All the nerves which reach the terminal part of the œsophagus from above the diaphragm.

III. If the operator removed only the gastric branch(es) which runs along the lesser curvature—

All the nerves to the cardiac orifice might escape destruction, except the branch from the hepatic plexus crossing between the layers of the lesser omentum. But in most cases even it would escape, as it would be above the lines of the omental incisions.

The problem remains, how best to produce sympathetic denervation of the cardiac orifice while doing as little damage as possible to other nerve structures. It may be said at once that any peripheral operation, to be successful, would have to be more radical than the procedure at present in vogue, because the gastro-œsophageal junction receives nerve-filaments from widely different sources, which

vary from case to case in relative size and importance. Theoretically, in order to be certain of securing complete sympathetic denervation it would be necessary to destroy: (1) The nerves accompanying the left gastric artery; (2) The branch from the hepatic plexus high up between the layers of the lesser omentum; (3) The left (and in some cases the right) inferior phrenic plexus; (4) Filaments from the left lumbar gangliated chain or from the terminal part of the left greater splanchnic nerve; (5) Thoracic œsophageal branches from the gangliated trunks and splanchnic nerves. Ultimately the relative importance of these nerve-supplies will be assessed by experimental methods, and then it will be possible to decide with certainty those which should be divided. At present, if any peripheral operation is considered, it would probably be advisable: (1) To remove only the middle and proximal parts of the left gastric artery, along with the surrounding fatty and nervous tissues—this would avoid damage to the œsophageal and gastric branches of the vagi, and would prove as effective as the more radical operation in destroying the sympathetic filaments; (2) To remove part of the left inferior phrenic artery and its associated nerves; (3) To divide any nerve-filaments passing across to the cardia from the hepatic plexus, high up between the layers of the lesser omentum. The procedure advocated by Recalde in 1932 of stripping the external muscular coat from the region of the cardiac sphincter and destroying the underlying Auerbach's plexus would be most effective in producing denervation, but both sympathetic and parasympathetic would suffer equally, and it could be argued that results achieved by this method might be due to mechanical rather than neurological causes.

Alternatively an attempt could be made to interrupt the nerve-fibres to the lower œsophagus as they lie in the gangliated trunk or rami. Many apparently leave the gangliated trunks between the 6th and 9th thoracic ganglia, and thus removal of these parts might interrupt the efferent sympathetic nerve-supply to the cardiac orifice. But it is impossible to dogmatize, for many of the efferent fibres might be conveyed in the greater splanchnic nerves, which often receive roots of origin from above the level of the 6th thoracic ganglia, and there must be a limit to the extent of operative interference, lest the remedy becomes more dangerous than the disease.

I am deeply indebted to Professor A. Low, Regius Professor of Anatomy, Aberdeen University, who has verified all anatomical observations by examination of the actual dissections, and to Professor J. R. Learmonth, Regius Professor of Surgery, Aberdeen University, for much valuable criticism and advice. The expenses of the investigation were defrayed with the aid of a grant from the Medical Research Council.

REFERENCES

ADAMSON, W. A. D., Case report on p. 872 of Knight's article (*see below*).

CRAIG, W. McK., MOERSCH, H. J., and VINSON, P. P., "Treatment of Intractable Cardiospasm by Bilateral Cervicothoracic Sympathetic Ganglionectomy: Report of a Case", *Proc. Staff Meet. Mayo Clinic*, 1934, ix, 749.

GASK, G. E., and ROSS, J. P., *The Surgery of the Sympathetic Nervaus System*, 1927. London: Baillière, Tindall & Cox.

HOVELACQUE, A., "Anatomie des Nerfs craniens et rachidiens et du Système grand sympathique chez l'Homme", 1927. Paris: Gaston Doin et Cie.

KNIGHT, G. C., "Sympathectomy in the Treatment of Achalasia of the Cardia", *Brit. Jour. Surg.*, 1935, xxii, 864.

KOLLMANN, J., " Ueber den Verlauf des Lungenmagennerven in der Bauchhöhle ", *Zeits. f. wissensch. Zool.*, 1860, x, 413.
KUNTZ, A., *The Autonomic Nervous System*, 1929. London : Baillière, Tindall & Cox.
LEARMONTH, J. R., " The Surgery of the Sympathetic Nervous System ", *Brit. Jour. Surg.*, 1937, xxv, 426.
LEARMONTH, J. R., Personal communication *re* successful case of sympathectomy for cardiospasm.
MCCREA, E. D'ARCY, " The Abdominal Distribution of the Vagus ", *Jour. of Anat.*, 1924, lix, 18.
RECALDE, *Arch. ital. di Chir.*, 1932, xxxii, 613 (quoted by KNIGHT).
WERTHEIMER, P., " L'Innervation et L'Enervation gastriques ", 1922. Lyon : Imprimerie-Express.
WHITE, J. C., *The Autonomic Nervous System*, 1935. London : Henry Kimpton.
WOOLLARD, H. H., " The Peripheral Sympathetic Nervous System ", *Brit. Jour. Surg.*, 1935, xxiii, 425.

EXTRADURAL HÆMORRHAGE*

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By the time a student graduates in this day and age, he will have heard a good deal about extradural hæmorrhage, a lesion which usually but not always arises from a rupture of the anterior branch of the middle meningeal artery (*Fig.* 249). Nevertheless the lesion is rare, and in a general practice a doctor is not likely to encounter more than one or two cases. If he is to have the satisfaction of saving these few patients he must keep in mind the usual syndrome and also appreciate that there are variations of this syndrome. In a large hospital centre his responsibility is usually one of diagnosis. In outlying districts he must on occasion be prepared to operate as there may not be time to call a consultant from a distance.

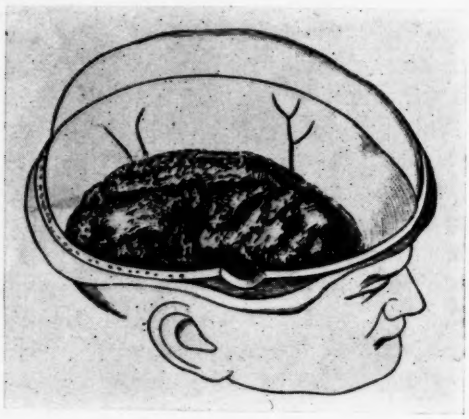

FIG. 249.—Middle meningeal hæmorrhage in a patient of Mr. Hilton's. (*Guy's Hospital Museum.*)

This paper is based on 20 cases of extradural hæmorrhage, the total number with which the author has had personal experience during the past ten years. Although the series is small, it illustrates many variations from the usual syndrom, and will serve as a basis for discussion. Indeed no excuse is needed for bringing the subject forward from time to time, as many cases continue to be undiagnosed and in certain instances surgical intervention is undertaken too late. Furthermore, I feel sure that taken by and large there is a high mortality in this group of patients —partly because there has been some other serious lesion associated with the extradural hæmorrhage, but also most of us can recall patients that would be alive to-day if there were two opportunities instead of one to treat the patient. Experience is a great teacher, but in this rare condition we must try to profit by the experience of others and not fail in the unusual case.

SUMMARY OF CASE REPORTS

Case 1.—Mr. Mc. Hit on back of head—never regained consciousness. Admitted to hospital two hours later, deeply unconscious and with Cheyne-Stokes respirations; P. 60; B.P. 220/110; bloody C.S.F.; bilateral Babinski and clonus, appearing first on right side; pupils moderately dilated, but equal. Bilateral exploration decided upon—small left-sided flap disclosed lacerated frontal and temporal poles. Right side not explored, because of patient's condition; died twenty minutes after leaving operating table. Autopsy disclosed fracture involving right temporal region and base and a large extradural clot; lacerated brain on left.

Comment.—When patients with extradural clot do not have a lucid interval there is usually an associated severe laceration of the brain.

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* Presented at the Cushing Society, Philadelphia, May, 1937.

Case 2.—Mr. J. Dazed; lucid interval of half an hour; fracture on side of lesion. Six hours after injury P. 62; R. 24; stuporous; not moving left side well. C.S.F. clear; homatropine in eyes. Twenty-four hours after injury P. 60–80; R. 24–32; coma. Two and a half days after injury P. 168; T. 104; R. 34 and stertorous; marked coma; marked left-sided paralysis; both pupils dilated; discs normal apart from filling of cups. Operation disclosed a large right extradural clot and linear fracture. Recovery after a critical period of 24 hours, when control of hyperthermia by colonic ice-water irrigation undoubtedly saved patient's life.

Case 3.—Mr. R. Dazed; lucid interval of half an hour. Two hours after injury P. 94; R. 36 and stertorous; B.P. 160/80; marked coma; no reflexes; no movement; right pupil large. Right-sided exploration 2½ hours after injury disclosed large right extradural clot; never regained consciousness; right pupil became small; wound opened and dura found next bone; died 12 hours after operation with mounting P., R., and T. Autopsy disclosed the lateral sinus lifted out of its bed by a backward extension of clot over the cerebellum.

Comment.—This patient's life could have been saved if the possibility of posterior extension over the cerebellum had been appreciated. When the posterior limit of the clot cannot be reached through the usual temporal approach, a second posterior bone opening should be made.

Case 4.—Mr. H. Dazed; thought to be drunk; lucid interval of 1 hour. Six hours after injury P. 72; R. 24, blowing in character; B.P. 125/75; deeply unconscious, but struggled when lumbar puncture was done, did not move right arm and leg well; dilated right pupil; bloody C.S.F. with pressure of 260 mm. Operation 6 hours after injury disclosed an extensive low-lying left extradural clot, a linear fracture, artery was bleeding freely at foramen spinosum; satisfactory convalescence and recovery.

Case 5.—Mr. MacF. Unconscious for possibly half an hour; lucid interval of 2 hours. Four hours after injury P. 50; R. stertorous; stuporous and restless. Twenty-four hours after injury in deep coma, not moving left side; right pupil large and fixed. Thirty-eight hours after injury P. 160; B.P. 60; deep coma; R. rapid. Operation 38 hours after injury disclosed large right extradural clot, linear fracture. Following operation condition critical for several hours until hyperthermia was controlled; developed pneumonia; eventual satisfactory recovery.

Comment.—Diagnosis and operation almost too late; efficient treatment of hyperthermia saved patient's life.

Case 6.—Mr. C. Dazed; lucid interval of 2 hours. Five hours after injury P. 60; R. stertorous; B.P. 160/108; comatose; left Babinski and clonus; left side not moved well; large right pupil; C.S.F. pink, with pressure of 300 mm. Five hours after injury admitted to hospital and taken to X-ray room; sudden respiratory failure and death before operation could be performed. Autopsy disclosed a large right-sided extradural clot.

Comment.—The patient should have been taken to the operating room on admittance to hospital. The diagnosis was clear without X-ray examination, and the seriousness of the patient's condition should have been appreciated.

Case 7.—Mr. S. Unconscious 5 minutes; lucid interval of 2 hours. Three hours after injury P. slow; R. quiet; B.P. 145; stuporous; pupils equal; lumbar puncture not done. Three and a half hours after P. 80; breathing stertorous; B.P. 160/110; comatose; left side not moved readily; reflexes not noted; right pupil dilated. Operation 4 hours after injury disclosed a large right extradural clot and linear fracture of the skull. Convalescence and recovery satisfactory.

Case 8.—Mr. C. Unconscious 3 minutes; lucid interval of 2 hours. One and a half hours after injury P. 54; R. quiet; B.P. 120/80; dull, but easily roused; no hemiparesis; pupils equal; C.S.F. clear, with pressure of 300 mm. Three and a half hours after injury, P. 50; R. stertorous; B.P. 160/65; deep coma; bilateral Babinski; both pupils widely

dilated, left had dilated first. Operation 3½ hours after injury. Following operation P. and T. remained elevated; the left hemiparesis persisted; patient never recovered consciousness. The wound was reopened and the dura found tightly against bone; death 5 days after operation. Complete autopsy did not account for the death.

Comment.—This case illustrates the urgency for operative interference in some of these patients. This patient could have been operated upon some two hours before when the diagnosis was still in doubt: an exploratory burr hole would have disclosed the clot, and at this stage recovery would have been certain.

Case 9.—Mr. B. Dazed; lucid interval of 2½ hours. Three hours after injury P. 45–50; R. stertorous; profound stupor; a large left pupil; C.S.F. pink. Six hours after injury P. 70–80; artificial respiration was necessary just as the operation commenced; B.P. 110; profound coma; moved both legs on painful stimuli; bilateral Babinski; large left pupil. Operation 6 hours after injury disclosed a large left extradural clot.

Comment.—Delay of another few minutes in this particular case would undoubtedly have resulted in death.

Case 10.—Mr. S. Dazed; lucid interval 2 to 4 hours. Twenty-four hours after injury P. very fast and thin; R. stertorous and fast; there was no response to painful stimuli; marked spasticity of all limbs; right pupil was dilated and was said to have been dilated 15 minutes after the accident. It was felt that patient was *in extremis* and there was probably a brain-stem hæmorrhage to account for the observation that the pupil had been dilated within a few minutes of the injury. Operation was not performed. Autopsy disclosed a large right extradural clot.

Comment.—This must have been an extremely rapid hæmorrhage to have produced a dilated pupil so soon after the accident. There may have been some direct injury to the third nerve. It was stated, however, that there was no hæmorrhage into the brain-stem.

Case 11.—Mrs. W. Dazed; lucid interval of 7 hours. Twenty-four hours after injury P. 160; rapid, irregular respirations; B.P. 140; profound stupor; no movement of the right arm and leg on painful stimuli; reflexes not noted; a large left pupil; C.S.F. clear. Operation 24 hours after injury disclosed a large extradural clot on the left side and linear fracture of the skull; satisfactory recovery after a critical post-operative period of several days.

Comment.—Case should have been diagnosed and operated upon earlier.

Case 12.—Miss B. Dazed; there was a linear fracture of the skull which was not seen because the wrong side of the head was next the plate; lucid interval of 12 hours. One hour after injury P. 90; had an epileptic seizure, following which the right pupil was dilated; C.S.F. was bloody. Twelve hours after injury R. quiet; no hemiparesis; pupils equal. Forty-eight hours after injury P. 120; rapid Cheyne-Stokes respirations; deep coma; bilateral Babinski; seemed to move sides equally on painful stimuli; right pupil slightly larger, did not re-act well to light. Operation 48 hours after injury disclosed typical right extradural clot lying rather high. The patient did not recover consciousness and died 6 hours after operation. Autopsy disclosed a ruptured liver; there was still considerable extradural clot, as oozing and bleeding had continued after operation.

Comment.—This patient was very critically ill at the time of operation; the case also illustrates the necessity of complete physical examination to determine the presence of traumatic injuries elsewhere in the body.

Case 13.—Mr. D. Dazed; hit back of head; lucid interval of 25 hours. Twenty-eight hours after injury P. 70; R. shallow; patient cyanosed; B.P. 40; marked coma; limbs flaccid; no deep reflexes; pin-point pupils; morphia had been given 3 hours before to control severe headache; C.S.F. bloody, pressure of 290 mm. Thirty hours after injury P. 64; colour improved; B.P. 100/50; marked coma. After 36½ hours P. 100; R. 22, with cyanosis; B.P. 130; responding slightly; limbs flaccid; pupils small, with some

nystagmus; C.S.F. bloody, with pressure of 310 mm. It was felt that the patient did not have a typical extradural hæmorrhage. Because of the respiratory failure following the morphia and because of some nystagmus a cerebellar exploration was decided upon; it was felt that possibly there was a large clot in the posterior fossa. A large extradural clot was exposed lying over the left cerebellar hemisphere and the left occipital lobe; it was extradural. Following the removal of this clot the patient's general condition improved considerably; however, he died on the day following operation. Autopsy disclosed moderate bruising of the right temporal lobe and a moderate amount of right subdural hæmorrhage. The site of operation was satisfactory, and the extradural clot had not reformed.

Comment.—An unusual place for a large extradural clot. If an earlier correct diagnosis could have been made, the patient's life would probably have been saved.

Case 14.—Dorothy D. Dazed after falling off a chair; lucid interval of 30 hours. Thirty-six hours after injury P. 100; R. quiet 25–30; roused with difficulty would take a drink; marked hypotonia, with easily elicited reflexes; pupils equal; C.S.F. clear; unable to make diagnosis. Forty-four hours after injury condition unchanged. While then being examined had an epileptic seizure, which definitely commenced on the right side of the body; it was followed by marked opisthotonos, sudden respiratory failure, and death. Autopsy disclosed an extradural clot completely covering the right cerebellar hemisphere; it was ¾ in. in thickness, there was a short 1-in. fracture radiating out from the rim of the foramen magnum on the side of the hæmorrhage. There was no evidence of injury to the brain elsewhere.

Comment.—I had never heard of extradural hæmorrhage over the cerebellum; the diagnosis was not even suspected. If the diagnosis could have been made operation would have been simple and recovery almost certain.

Case 15.—Mr. R. Unconscious 5 to 10 minutes; lucid interval of 48 hours. Three to four hours after injury P. 80; R. quiet; well orientated; no paresis; pupils equal; C.S.F. clear, pressure of 300 mm. Twenty-four hours after injury P. 65–70; R. quiet; restless and drowsy; no paresis; pupils equal. Forty-four hours after injury P. 60 to 65; R. quiet; restless, drowsy, no movement left side; bilateral Babinski; dilated right pupil. At time of operation, 50 hours after injury, condition very much the same; large right-sided extradural clot.

Comment.—Case showed a very gradual march of signs and symptoms.

Case 16.—Mr. F. Unconscious 5 minutes; lucid interval of 48 hours. Thirty hours after injury P. 60 to 65; R. quiet; B.P. 130/70; dull but responding well; no paresis, but bilateral Babinski; dilated right pupil; lumbar puncture not done; definite choking of discs. Forty-six hours after injury general condition unchanged, but a marked paresis of left side had developed; named test objects readily in either hand. Forty-eight hours after injury sudden respiratory failure; rushed to operating room. Under artificial respiration and unsterile set-up a large right-sided extradural clot was removed; on operating table brain became extremely tight, dura was opened through an enlarged bone opening, the cortex ruptured; respirations commenced; condition again critical a few hours later; cerebellar decompression done and a marked herniation of the cerebellum uncovered; improved for few hours, then death.

Comment.—This case is the type with a long lucid interval, but illustrates the point that such cases may suddenly become very critical and even though the clot is removed recovery may not take place.

Case 17.—Mr. K. Unconscious for 7 hours; possibly intoxicated during this period; lucid interval of 3 days. Forty-eight hours after injury P. 60; dull, but clear; C.S.F. slightly bloody. Condition 72 hours, 5 days, and 8 days after injury very much the same. On the 8th day, however, the C.S.F. pressure was only 75 mm.; there was questionable choking. On the 11th day P. 50; R. quiet; difficult to rouse; some jerking of right shoulder; pupils equal. Exploratory burr hole made on the left side with negative findings; on the right

a large extradural clot covering the parietal lobe was removed. Recovery was satisfactory and uncomplicated.

Comment.—The lateralizing diagnosis in this case was complicated by the fact that the patient's left leg had been amputated; also there was an unusually long lucid interval.

Case 18.—Miss H. Dazed a few moments only; lucid interval of 11 days; patient was still quite responsive and co-operative at the time of operation. Nine days after injury P. 100; R. quiet; B.P. 130/90; very responsive, but aphasic; a flaccid right-sided paralysis; reflexes not as brisk as on the left; Babinski on the right; diminished sensitization for pain over the paralysed side; pupils were small, equal, reacted to light; eye movements were full, slight choking of the discs. Operation disclosed a very large extradural clot on the left side covering most of the hemisphere as well as elevating the temporal lobe from the floor of the skull. When the clot was removed a bleeding-point was not disclosed; recovery was satisfactory.

Comment.—This was one of the largest clots in the series and bleeding must have taken place very slowly to have produced the marked paralysis without any disturbance of consciousness, pulse, and respirations.

Case 19.—Mrs. G. Unconscious or very stuporous for 3 hours; a lucid interval of 11 days, when the patient continued to respond well until time of operation. Four days after injury P. 65–70; R. quiet; B.P. 124/78; responded well; no paresis; pupils equal; C.S.F. pink, pressure of 300 mm.; discs clear. Nine days after injury P. 65–70; R. quiet; responding, but dull; a right Babinski and clonus; 2-point sensation was normal; left pupil larger than right, questionable choking. Operation 11 days after injury disclosed a large extradural clot practically all above the Sylvian fissure; the brain was not under tension at operation. Recovery was satisfactory and uncomplicated.

Comment.—Persistent, severe headache, a linear fracture, and the development of a Babinski and clonus led to operation on this patient.

Case 20.—Mr. S. P. Dazed, possibly intoxicated; a lucid interval of 21 days. Six hours after injury there was no record of the patient's P. and R., but there was marked hemiparesis and aphasia. Three weeks after injury P. 50–70; R. quiet; B.P. 160/90; dull, responded readily, aphasic; right-sided hypotonia; right Babinski; right pupil slightly larger than the left; C.S.F. yellow, with a pressure of 175 mm.; slight choking of the discs. Operation 3 weeks after injury disclosed a large extradural clot lying over the parietal lobe.

Comment.—The clot was nearly missed by the usual temporal burr hole; it is quite possible that intoxication saved this patient's life by preventing excessive movement and a consequent increase of bleeding after the injury.

THE SYNDROME

In 1886, Jacobson[1] published a long article of one hundred and fifty pages based on a study of 70 cases. This paper is filled with sound clinical and pathological observations. From this study the typical or average clinical syndrome emerged, and has come down through the years in our standard text-books of surgery. Two additional diagnostic measures were introduced toward the close of the last century: (1) Lumbar puncture to determine intracranial pressure and the presence or absence of blood in the cerebrospinal fluid;* (2) The detection of a fracture crossing the middle meningeal artery or its branches by X-ray examination.†

Briefly the syndrome which is impressed on medical students to-day is as follows: An individual is dazed or rendered unconscious for a few minutes by

* Quincke suggested lumbar puncture in 1891.

† X rays were discovered by Roentgen in 1895.

a blow on the head. Usually recovery occurs without the development of serious symptoms. Such patients, however, should be kept under close observation and the possibility of extradural hæmorrhage considered. If a clot is forming in the extradural space it usually causes signs of pressure within an hour or two. The most important evidence of this pressure is a dulling of the mental faculties, which steadily progresses to coma, with stertorous breathing. Other signs such as slowing of the pulse and raising of the blood-pressure are important, but frequently there is no record of such changes, because observation had not been possible or accurate during the early period when these signs frequently are present only for a short time. As coma deepens, the respiration, pulse, and temperature rise; these changes indicate failure of the medullary centres to respond in the normal physiological way to a rapid increase of intracranial pressure. They are very serious signs, and where possible, operation should be performed before they develop. During the development of the stupor, examination discloses three lateralizing signs :—

1. Œdema of the scalp in the temporal fossa, suggesting a linear fracture. This can be confirmed by X-ray examination. If the fracture crosses the groove of the middle meningeal or one of its branches, it is of special significance, as almost always the lesion is produced by a fracture tearing this artery.

2. A dilated fixed pupil on the side of the lesion. This is due to displacement of the brain, which causes stretching and pressure on the third nerve. It is one of the most constant and valuable lateralizing signs.

3. A positive Babinski and diminution of active movement of the arm and leg on the side opposite to the lesion—that is, on the side opposite to the dilated fixed pupil. This is due to pressure by the large clot on the underlying cerebral hemisphere. The hemiparesis often has to be elicited by painful stimuli because the patient is usually stuporous when it develops.

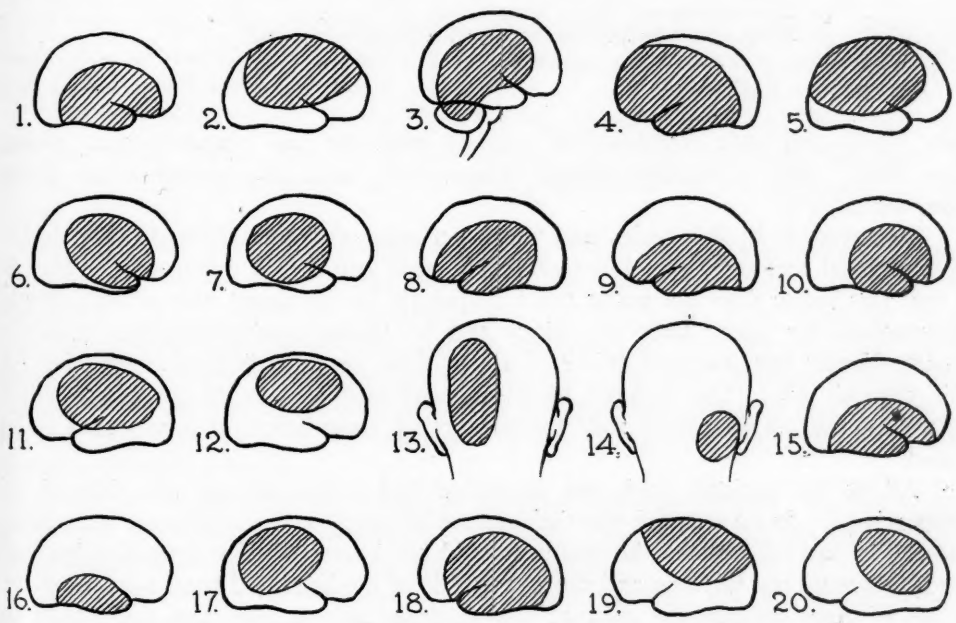


FIG. 250.—Showing the approximate size and situation of the extradural clots in *Cases* 1–20.

In the presence of the above syndrome, lumbar puncture discloses a high degree of pressure. If the fluid is clear or only slightly bloody the increased pressure is almost certainly due to an extradural hæmorrhage. On the other hand a very bloody cerebrospinal fluid indicates some degree of cerebral damage even if there is an extradural clot. I have operated upon several such patients whose syndrome differed in no way from that outlined, except that the lucid interval was less well defined and the progress of the syndrome much more rapid. Operation usually disclosed a small amount of extradural blood, but the main lesion was a massive bruising of the fronto-temporal lobes. In my experience practically all of these patients have died, and autopsy disclosed a bilateral injury to the brain, more marked on the side of the dilated pupil.

In the 20 patients considered the main lesion was a massive extradural clot; where death occurred this lesion was considered to be the cause of death, although autopsy showed some cerebral contusion in several instances, and in one patient a ruptured liver. (*Fig.* 250.)

Initial Injury and Fracture (*Table I*).—The initial clinical evidence of injury to the brain was insignificant in 18 of the 20 patients; 11 patients were dazed, 7 unconscious for only a few minutes; 1 was unconscious or very stuporous for

Table I.—Initial Clinical Findings in Extradural Hæmorrhage

	Cases
Fracture on side of lesion	19
No fracture	1
	20
Dazed	11
Unconscious a few minutes	7
Unconscious or dazed three hours	1
Deeply unconscious two hours, with no lucid interval ..	1
	20

three hours and then continued to respond until she was operated upon eleven days later; only 1 remained deeply unconscious until the operation two hours after the injury.

The records in this series and the experience of others show that the lesion of extradural hæmorrhage follows relatively mild concussion syndromes. Why is it that this lesion does not occur more frequently in the group who remain deeply unconscious for hours after the injury and on whom lumbar puncture shows a grossly bloody cerebrospinal fluid? I know of no very satisfactory explanation for this clinical fact. The lowered blood-pressure and œdema of the brain which occurs in the severe injury may promote rapid coagulation in a ruptured dural vessel.

All of the patients with one exception had a fracture on the side of the hæmorrhage. In no instance was the fracture bilateral, so it was of great lateralizing value. In several patients the fracture could not be seen before operation because the plates were not bilateral and the wrong side of the head had been next the plate. In all of these patients the fracture was disclosed at operation and could then be seen in the plates when one knew that it was present. In two patients the fracture

was not seen because of its unusual position, involving the wing of the sphenoid in one instance and the occipital bone in the other.

In *Case* 13, where the hæmorrhage was over the occipital lobe and cerebellum there was no fracture seen in the radiograph or on careful search at autopsy. Jacobson mentions several similar cases. Keith[2] has had a case where the hæmorrhage was in the usual temporal position and fracture could not be seen at autopsy or by X-ray examination. These cases are so rare that the absence of a linear fracture in good bilateral radiographs is strong evidence against the diagnosis of an extradural hæmorrhage. It must not be forgotten that when X rays are not available, careful palpation will disclose œdema of the scalp over a linear fracture.

The Lucid Interval (*Table II*).—Nineteen patients had well-defined lucid intervals. Until a few years ago I had felt that a lucid interval longer than an hour or two was most unusual. Probably the most important and significant feature in this series is the number of patients who had prolonged lucid intervals. As a matter of fact the diagnosis was not made or suspected in one or two of our patients because of this. *Table II* shows that 10 patients had relatively short lucid intervals. An equal number had intervals varying from seven hours to twenty-one days. This group of ten patients constitutes an important variation from the text-book description. In a larger group of patients the incidence of delayed cases or prolonged lucid intervals would probably be considerably less than 50 per cent as in this series. It is important at least to suspect the diagnosis in this group, as in two instances the patient became critically ill very rapidly after remaining in good shape for several days.

In one patient, *Case* 1, there was no lucid interval. Autopsy disclosed a marked bruising of the brain as well as a large extradural clot. It is not likely that many such patients recover after operation. Dr. van Wagenen, of Rochester, N.Y., informs me that he has recently had such a case. The patient was hit with a golf ball. There was deep unconsciousness without a lucid interval. Recovery followed operation, which disclosed a large extradural clot as well as marked bruising of the underlying temporal lobe.

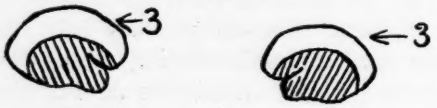
The Dilated Pupil (*Table II*).—Fifteen patients had a dilated pupil on the side of the lesion. Usually it was marked, and associated with complete or partial loss of reaction to light and a varying degree of loss of movement of the eyeball.

In 5 patients this valuable sign was absent. This was to be expected in *Cases* 13 and 14, where the hæmorrhage was not in the usual position over the cerebral hemisphere. In 2 others the syndrome was not acute, the lucid intervals being three and eleven days. In the fifth patient, there was no lucid interval—and there was moderate dilatation of both pupils. There is a good explanation for the absence of the sign in this patient, as there was sufficient bruising and œdema of the temporal pole on the side opposite to the hæmorrhage to prevent lateral displacement of the brain.

In the acute cases the dilated pupil develops with the stupor. In this series the shortest time after the accident was two hours. In the delayed or chronic cases it may appear after days, and then not be very marked.

The explanation of the dilated pupil in cranio-cerebral injuries has interested us. Theoretically there are several explanations. We have been able to make the following observations :—

Table II.—DETAILS OF *Cases* 1—20

NUMBER OF CASES		DILATED PUPIL	LUCID INTERVAL	DEATHS
1		0	0	1
2		2	½ hr.	1
1		1	1 hr.	
6		6	2–4 hr.	3
1		1	7 hr.	
1		1	12 hr.	1
1	OCCIPITAL AND CEREBELLAR	0	25 hr.	1
1	CEREBELLAR ONLY	0	30 hr.	1
2		2	48 hr.	1
1		0	3 d.	
2		0 1	11 d.	
1		1	21 d.	
20		15		9

1. I am indebted to Dr. Linell for *Fig.* 251. This patient died three hours after an injury. The dilated right pupil was explained by the hæmorrhage in the right side of the brain-stem.

2. *Fig.* 252 shows the right third nerve pulled out of the brain-stem and a definite bruising of the left nerve where it is in contact with the posterior cerebral artery. This patient died two hours after a severe injury.

The above two observations offer a pathological basis to explain a dilated pupil which is observed within a few minutes of the accident. In *Case* 10, of the extradural series, the dilated pupil was said to have been present fifteen minutes

FIG. 251.—Sections from left and right sides of the brain-stem, showing definite hæmorrhage on the right side. This caused a dilatation of the right pupil. (*Dr. Linell.*)

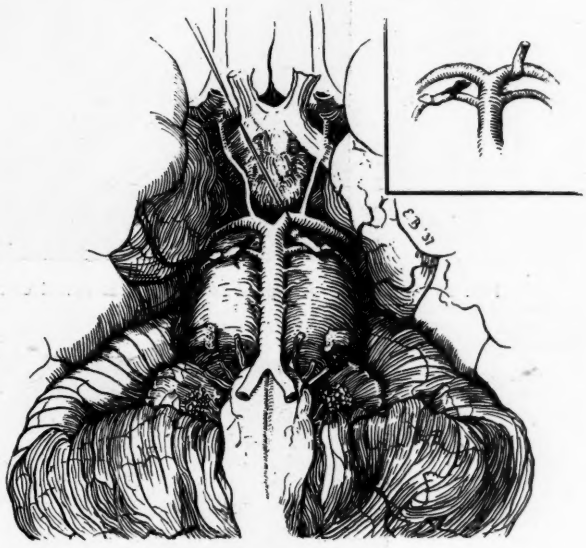


FIG. 252.—Trauma to oculomotor nerves (*see* text).

after the accident; there was probably some such lesion along with the extradural hæmorrhage. The autopsy was not done with sufficient care to determine this point.

3. The third observation, which in my opinion probably explains the majority of the cases under consideration in this paper, was made at the autopsy on *Case* 6. *Fig.* 253 shows the large extradural clot on the right side.

It was perfectly apparent that in this patient the right third nerve was pulled around the corner of the clivus of the sphenoid bone by the upper part of the brain-stem being pushed to the left by the clot. This mechanism is shown in *Fig.* 254. At point X there was a definite groove on the under side of the nerve. *Fig.* 255 shows a photograph and microscopical section of the nerve, and demonstrates a groove in the nerve. We felt that it was pressure at this point which interfered with the function of the nerve and caused the dilated pupil.

4. The fourth observation has to do with herniation of the temporal lobe through the ring of the tentorium. *Fig.* 256, from Linell's collection, illustrates such a condition, and shows how the function of the third nerve may be

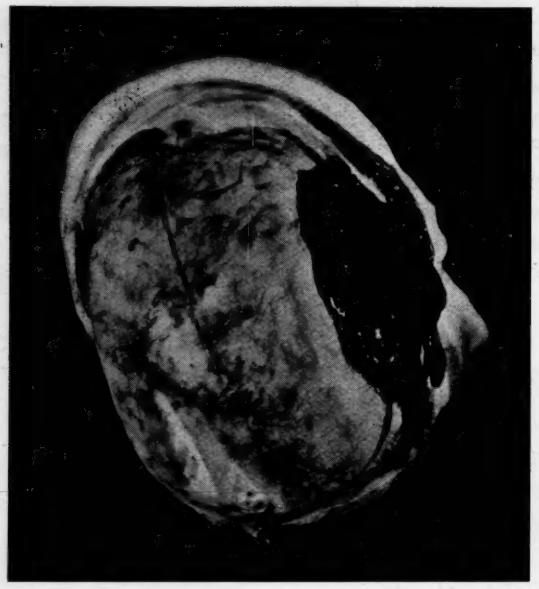


FIG. 253.—*Case* 6. A typical large right extradural clot causing dilatation of the right pupil.

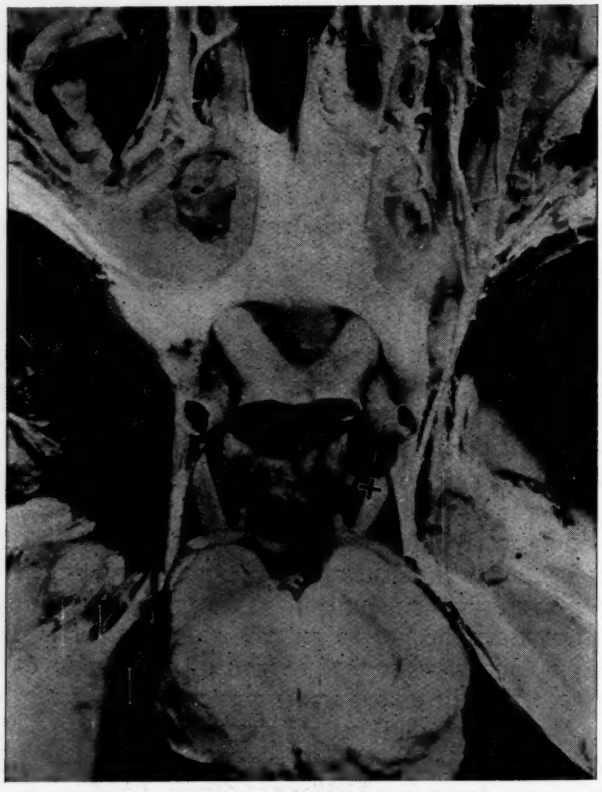
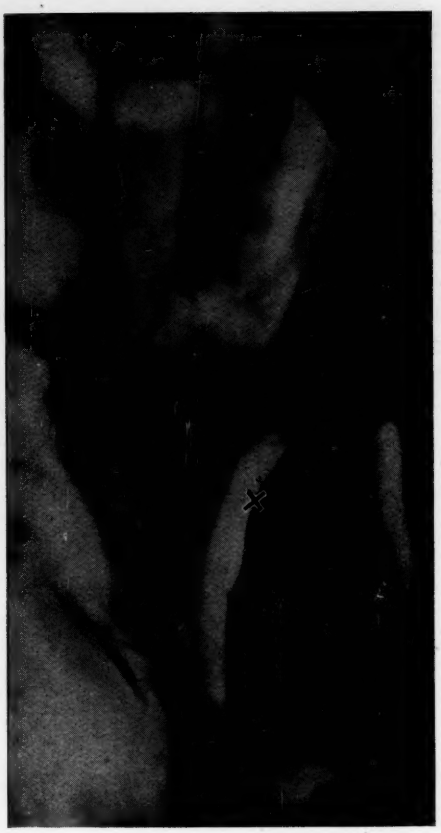


FIG. 254.—Showing a definite groove at X on under side of the nerve, adjacent to the clivus of the sphenoid.



A

B

FIG. 255.—A, Photograph showing a groove in the 3rd nerve at X, produced by pressure from the clivus of the sphenoid bone; B, Microscopical section of the nerve.

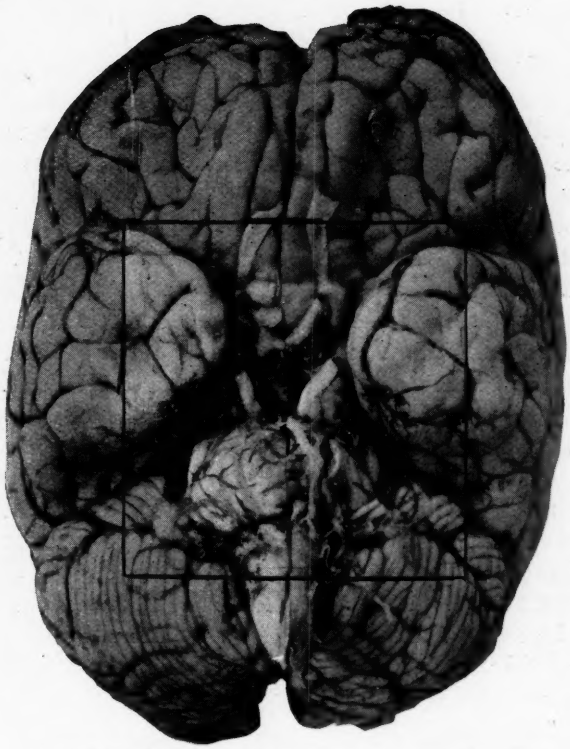


FIG. 256.—Bilateral herniation of the hypocampal uncus at X.

diminished by this mechanism. In this particular patient, the primary lesion—a large bilateral frontal-lobe meningioma—had produced a high degree of intracranial tension of long standing. This herniation may occur with acute intracranial tension produced by injury, but so far I have not been able to observe this herniation as an explanation for third-nerve palsy in cranio-cerebral injuries.

Lumbar Puncture and the Cerebrospinal Fluid (*Table III*).—Lumbar puncture was not done on 6 patients. In 9 cases there was blood in the fluid. In 4 there was a sufficient quantity to postulate considerable damage to the brain. Three of these patients died, and autopsy showed gross bruising of the brain. In 5 patients blood was present in small amounts. One of these patients died because he was not operated upon, and autopsy showed no gross bruising of the brain. In 5 patients the fluid was clear; 2 of these died—namely, *Case* 14, where the lesion was over the cerebellum, and *Case* 8, where operation was too late.

Table III.—The Cerebrospinal Fluid in Extradural Hæmorrhage

No. of Cases	*Blood in the Cerebrospinal Fluid*	*Deaths*
6	Not investigated	–
4	Considerable blood	3
5	Small amount of blood	1
5	No blood	2
20		
	Pressure of the Cerebrospinal Fluid	
11	Not recorded	
7	High—290–300 mm.	
2	Low—75–175 mm.	
20		

The pressure was recorded in 9 patients. It was high, in the neighbourhood of 300 mm., in 7 patients. In 5 of these the pressure was recorded six hours after the injury; in the other 2 within twenty-four hours. In two patients, *Cases* 17 and 20, where the pressure was low, it was recorded eight days and twenty-one days respectively after the injury.

In general it may be stated that a considerable amount of blood in the cerebrospinal fluid indicates bruising of the brain, and consequently the mortality-rate is higher in this group. Furthermore, the diagnosis is somewhat in doubt where there is considerable blood, as patients who have extensive bruising of the brain may present the cardinal diagnostic features of extradural hæmorrhage. On the other hand, in the presence of cardinal signs the diagnosis is practically certain where the fluid is clear or only slightly pink.

It cannot be stated too emphatically that these punctures should be purely diagnostic, and in no suspected case should more than a few cubic centimetres be withdrawn, for fear of converting a reasonably good risk into a poor one by producing a cerebellar herniation. I can recall at least one such case.

In the acute cases a high pressure should be the rule. In the delayed syndrome the pressure may be below normal, as in *Cases* 17 and 20.

Disc Changes (Pre-operative) (*Table IV*).—In 13 patients the condition of the disc is not recorded. It is safe to assume, however, that in most of these patients

there was no striking change in the disc. It is unlikely that any significant disc changes occur within the first twelve hours. In *Case* 16 there was definite choking thirty hours after the injury. In 5 patients, ranging from two and one-half to twenty-one days after the injury, there was slight or questionable choking. By this I mean changes that were not very definite, and they might have been normal for that particular patient.

Table IV.—PRE-OPERATIVE DISC CHANGES IN EXTRADURAL HÆMORRHAGE

No. of Cases			
13	Not recorded		
1	Normal		
	Time after Injury		
	Hours	Days	
1		2½	Cups filled
1	30	—	Definite choking
1		11	? choking
1		11	Slight choking
1		9	? choking
1		21	Slight choking
20			

Patients who survive for a prolonged interval after the injury probably do so because the bleeding is slow and the intracranial contents become adjusted to the space-taking hæmorrhage, and intracranial tension is not materially raised; in fact, it may be low. In *Case* 13, five days after the injury, the spinal pressure was 125 mm.; three days later it was 75 mm. Yet the clot in this case was at least 1 in. thick and 3½ in. in diameter. In *Case* 16 there was a similar clot, and three weeks after the injury the pressure was 175 mm., with slight choking.

In a number of cases, especially the ones of long standing, there was no tendency for the brain to expand after the clot was removed—further evidence that the brain itself was not under tension.

We may conclude that in the early acute cases where intracranial pressure is high there is not time for the disc changes to develop. Our earliest case of definite choking was thirty hours after the injury. In the chronic or delayed cases intracranial tension becomes adjusted and disc changes are insignificant.

THE TIME RELATION OR MARCH OF THE IMPORTANT SIGNS IN THE SYNDROME

For various reasons it is difficult to get accurate observations on the march of the syndrome. *Table V* is a composite one, constructed from a study of these twenty patients, and represents, I believe, the march of symptoms and signs which would have been seen if the patients had been accurately observed from the time of the accident.

The syndrome may be extremely rapid, with little spacing of the signs, as in *Case* 3, where the patient was *in extremis* two and one-half hours after the injury. In other patients the syndrome does not advance beyond dulling of the mentality, slowing of the pulse, paresis, and possibly some dilatation of the pupil, as in *Cases* 17, 18, 19, and 20.

Table V.—The Time Relations of the Important Signs in the Syndrome

Early Signs.—
1. Dulling of the mental faculties and slowing of the pulse
2. A positive Babinski
3. Paresis
4. Dilating pupil
5. Definite and obvious stupor

Serious Signs.—
1. Stertorous respirations
2. Cheyne-Stokes respirations
3. Profound coma
4. Rising pulse
5. Bilateral Babinski
6. Falling blood-pressure
7. Complete lack of movement or reflexes

DIAGNOSIS OF EXTRADURAL HÆMORRHAGE OVER THE CEREBRAL HEMISPHERE

The typical text-book syndrome has been outlined at the commencement of this paper. About half of the cases in this series fit in with this description. The variations in the usual syndrome have been statistically considered under various headings, such as Initial Injury; Lucid Interval; The Dilated Pupil; Cerebrospinal Fluid; Disc Changes. The outstanding variation which has impressed me is the prolonged lucid interval which occurred in 10 of the 20 patients. The absence of a lucid interval is rare—and these patients will probably show severe concomitant brain bruising, so that recovery is unlikely.

The timing of signs is shown where possible in *Table I*, and also considered under the heading "The Time Relation or March of the Important Signs in the Syndrome".

DIAGNOSIS OF EXTRADURAL HÆMORRHAGE OVER THE CEREBELLUM

Fig. 257 shows an extradural clot $\frac{3}{4}$ in. thick and 2 in. in diameter, which was situated over the right cerebellar hemisphere in *Case* 14. The only other autopsy finding was a 1-in. fracture involving the occipital bone and running into the foramen magnum on the same side as the hæmorrhage. There was no injury to the brain and no cerebellar herniation. This patient, a child, had a trivial injury, a lucid interval of thirty hours—the cerebrospinal fluid was clear. The fracture could not be seen in the radiograph. The pupils were equal; there was no lateralizing weakness of the limbs or disturbance of reflexes; there was no nystagmus, although there might have been earlier, with better co-operation. The patient died suddenly from respiratory failure forty-four hours after the injury and fourteen hours after the onset of stupor occurred. Looking back on the case I can still vividly recall the marked flaccidity of all the limbs. This one case of extradural hæmorrhage situated only over the cerebellum is probably a pathological curiosity. However, I have considered it worth while to devote this short paragraph to the diagnosis of extradural hæmorrhage over the cerebellum in the hope that some day

an acute clinician will diagnose such a condition. Certainly this patient's life could have been saved.

Two other patients, *Cases* 3 and 13, are of interest under this heading. In *Case* 3, the hæmorrhage was from the middle meningeal artery; the lucid interval lasted only half an hour, indicating the severity and rapidity of the hæmorrhage. At operation a large clot covering the cerebral hemisphere was removed. Autopsy twelve hours later showed an extension of the clot back over the cerebellum. This had not been recognized at operation. Since that experience I have always made a second opening well back in the skull if it is impossible satisfactorily to determine the posterior extent of the clot from the usual temporal exploration.

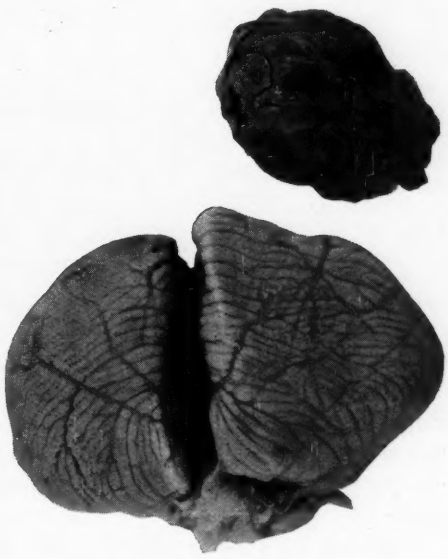


FIG. 257.—*Case* 14. An extradural clot over the right cerebellar hemisphere.

Case 13 was complicated by the patient being brought into hospital at the point of death from respiratory failure, which developed rapidly after $\frac{1}{4}$ gr. of morphia had been given to control severe spasms of headache. The correct diagnosis and operative procedure was carried out. The clot over the occipital lobe and cerebellum was evacuated; later a cerebellar decompression was done to relieve a pressure cone. Autopsy showed a moderate *contre-coup* bruising of the temporal and frontal poles. There was no recurrence of the extradural clot. The patient would probably have made a satisfactory recovery if he had not been so critically ill at the time of operation. In this case, morphia and not lumbar puncture precipitated a catastrophe.

A CONSIDERATION OF THE DEATHS

Nine patients died in this series—a mortality-rate of practically 50 per cent. The most certain and obvious cause of death was failure to operate; there were three such cases. (*Table VI.*)

Table VI.—CAUSES OF DEATH IN FATAL CASES

	Cases
Failure to operate	3
Concomitant severe injury	2
Extension of clot over cerebellum	1
Delay in operation	3
	9

Failure to Operate (3 Cases).—

Case 6 died of respiratory failure half an hour after being admitted to hospital. The diagnosis was made, but the patient died of respiratory failure while radiographs were being made. He should have been taken to the operating room and not the

X-ray room. The house-surgeon involved now appreciates the urgency of surgical intervention in some of these patients. I had had to learn very much the same lesson in *Case* 8. In such matters as these it is difficult to learn from the experience of others.

Case 10 was thought to be *in extremis* when seen in the country. It was also thought that he had a brain-stem lesion because of the marked spasticity of all limbs, and the history that there was a dilated pupil fifteen minutes after the accident. He lived on for another six hours, and might have been saved by operation.

Case 14. In this unusual case the presence of an extradural clot over the cerebellum was not even suspected. Operation would have been simple if the diagnosis had been made.

Death because of Concomitant Severe Injury (2 Cases).—

Case 1. With no lucid interval, there was a marked bruising of the temporal lobe as well as the extradural hæmorrhage.

Case 12. Treatment was undertaken late. Apart from the remains of some extradural clot, autopsy disclosed bruising of the brain and a ruptured liver.

Unrecognized Extension of the Clot down over the Cerebellum (1 Case).—

Case 3. This patient might have been saved if the extension of the clot downwards over the cerebellum had been recognized at the time of operation. Since this experience I have made a practice of making a second burr hole well back if the posterior extent of the clot cannot be determined through the usual temporal approach.

Delay in Operation (3 Cases).—

Case 8. Two hours after the injury the lesion was suspected—one and one-half hours later he was *in extremis*. He lived for some five days after operation, but never recovered consciousness. Autopsy showed no cause for death—there was no recurrence of clot.

Case 16. Patient was in good shape forty-eight hours after the injury; he then had a sudden respiratory failure. Operated on under artificial respiration, the clot was removed; the brain was very tight and a large decompression was made—slow respirations commenced, but no recovery of consciousness. Cerebellar decompression uncovered a marked cerebellar herniation; death followed a few hours after this procedure.

Case 13. An unusual case, with hæmorrhage over the occipital lobe and cerebellum; no fracture; lucid interval of twenty-five hours, followed by spasms of severe pain in head associated with opisthotonus; sudden respiratory failure after $\frac{1}{4}$ gr. of morphia; some recovery; clots removed by operation, but patient never recovered consciousness and died forty-eight hours after operation. Autopsy showed some *contre-coup* bruising of the frontal and temporal lobes, but earlier operation before the morphia would probably have saved the patient.

Such experiences as these make one feel that exploratory burr holes should be made as soon as there is any reasonable suspicion of a diagnosis of extradural clot. Theoretically 7 of these 9 deaths might have been averted by better diagnosis and earlier operation. It is also true that a number of the patients who recovered could have been operated upon much earlier and their recovery made much more certain.

DIFFERENTIAL DIAGNOSIS

Bruising and Œdema of the Brain.—Bruising and œdema of the brain may give the cardinal signs and symptoms of extradural clot. In the last analysis the only sure diagnostic measure is exploration. If the bilateral burr holes are negative no harm will have been done.

Intracerebral Hæmorrhage.—In two of the delayed cases in this series a diagnosis of intracerebral hæmorrhage was made because a profound hemiplegia came on within six hours of the injury yet the patients continued to respond. We argued that an extradural clot large enough to produce such a marked hemiplegia within a few hours would have produced a marked coma; however, such was not the case in these two patients. One patient, who had a localized intracerebral hæmorrhage involving the right temporal lobe, was successfully operated upon. Operation was performed ten days after injury because of stupor, choked discs, a left homonymous field defect, and slight hemiparesis. In my experience extradural or subdural hæmorrhage does not cause defect in the visual field.

Subdural Hæmorrhage.—The two successful cases of acute subdural hæmorrhage that I have operated upon have each had a long lucid interval and then developed increasing headache, stupor, and spinal fluid pressure. They did not have a dilated pupil and hemiparesis was not marked. They were operated upon in from ten days to two weeks after the injury, and in each instance the lesion was bilateral.

TREATMENT

The Operation.—The whole head is shaved and atropine but no morphia is given. If the patient is in a deep coma local infiltration between the scalp and the bone suffices. Usually a general anæsthetic is desirable for two reasons: (1) These

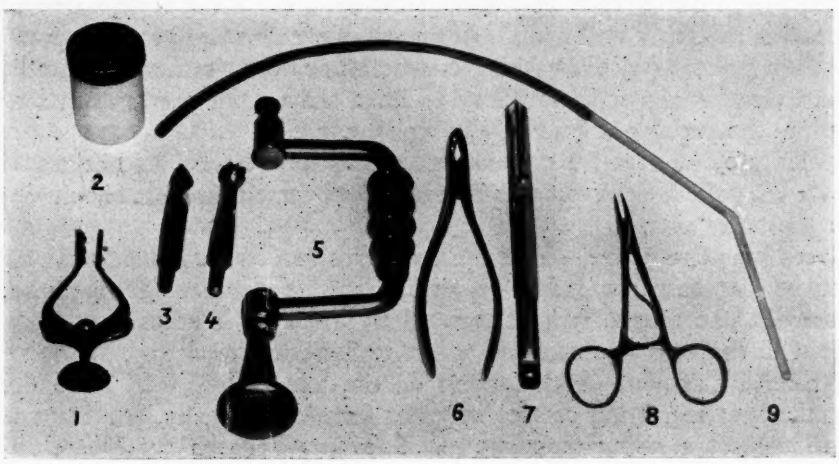


FIG. 258.—Special instruments: 1. Self-retaining retractor; 2, Bone wax; 3, 4, Hudson drills; 5, Hudson drill handle; 6, Rongeurs; 7, Silver clip holder; 8, Silver clip forceps; 9, Sucker.

patients are apt to become very restless and non-co-operative; (2) A general anæsthetic causes an increased congestion and tension in the brain, which is a great aid in bringing the dura up against the bone after the clot has been removed.

In administering the general anæsthetic, whether it be ether, chloroform, or ether and chloroform, it is most important to maintain a free airway. Gas is not a desirable anæsthetic in these cases.

In addition to a good spot-light, the special instruments and equipment that are desirable and necessary properly to perform the operation are shown in *Fig.* 258.

TECHNIQUE.—A short 1-in. incision is made just above the upper and anterior tip of the ear. The bone is exposed by splitting the temporal muscle; a linear fracture is usually seen at this stage. A self-retaining mastoid retractor is inserted. The bone is perforated by a Hudson burr or circular trephine. If the diagnosis is incorrect the dura is found adjacent to the bone.

If a clot is exposed, the operation is proceeded with by enlarging the bone opening to at least 1½ in. in diameter. The clot is removed by suction or the finger. Usually a spurting meningeal artery can be located in the exposed dura. To gain access to this artery or to remove all the clot it may be necessary to enlarge the bone opening still farther. The bleeding vessel is controlled by a ligature carefully passed so as not to injure a vessel in the underlying brain. A silver clip, applied and left in position, is usually a more efficient and easier method of controlling the vessel. The vessel may be torn very low in the temporal fossa, possibly right at its entrance into the skull through the foramen spinosum; in such a situation the foramen may be plugged with cotton, or the vessel coagulated. Not infrequently one fails to see a definite spurting vessel; this is especially true in the delayed cases. A general ooze from bone and dura is frequently a troublesome feature even in the cases where the main meningeal vessel has been controlled. This is especially so in the delayed cases where the brain is not under tension and does not expand and hold the dura against the bone after the clot is removed. Gentle packing with gauze usually controls oozing. Occasionally it is desirable to make extra burr holes and to suture the dura up to the scalp through these openings. Unless the dura comes up snugly against the bone and there is little or no oozing, the wound is left fairly well open. At a subsequent dressing in the operating room twenty-four hours later, the packing can be all or partly removed and the wound sutured. Occasionally it is necessary to leave the wound partly open for several days, while the packing is gradually being removed.

In the case of a negative exploration a second burr hole should be made farther back over the parietal area. If this is negative the same procedure should be carried out on the other side.

Post-operative Care.—The patient is put to bed on his side and the head is placed so that the airway is free—if respirations are stertorous this is of the utmost importance. The tongue is kept forward by a suture if necessary. Tubes may be placed in the nose or inside the cheek to increase the airway.

A careful half-hour check is kept on the temperature. In the seriously ill patient the heat regulating centres may not function normally, and steps must be taken to control a rising temperature. Sponging may be sufficient; a rectal irrigation with ice water for a temperature above 104° is most efficient and often necessary—a ten- or twenty-minute irrigation will quickly lower a temperature; it may have to be repeated frequently. The control of hyperthermia is definitely a life-saving measure in certain cases.

Re-exploration is indicated if one suspects that the clot has re-formed; apart from this there is little else of specific importance, except the maintenance of a free

airway and the control of hyperthermia; all of these three measures are essential in certain patients.

SUMMARY AND CONCLUSIONS

1. Extradural hæmorrhage is an infrequent lesion.

2. A lucid interval is the most valuable diagnostic sign. It may occasionally be absent.

3. A prolonged lucid interval is not infrequent and may confuse the diagnosis. These cases may very rapidly become critically ill.

4. A dilated pupil is a very valuable diagnostic and lateralizing sign. It may occasionally be absent.

5. Paresis is a valuable diagnostic and lateralizing sign. It may be difficult to establish in a stuporous patient. It may also be a false localizing sign and be on the same side as the lesion, in which case it is usually caused by *contre-coup* bruising of the hemisphere on the side opposite to the hæmorrhage.

6. Clear or faintly blood-tinged cerebrospinal fluid in the presence of a lucid interval followed by stupor makes the diagnosis almost certain; on the other hand if the fluid is very bloody the diagnosis is in doubt, as the syndrome may be produced entirely by the bruising or laceration of the brain.

7. A fracture is a valuable diagnostic and lateralizing sign, but it may be almost impossible to see when the fracture is not next to the X-ray plate; careful examination discloses œdema of the scalp overlying a fracture. Cases do occur without a fracture.

8. Extradural hæmorrhage is a serious lesion. The high mortality-rate impresses one with the necessity for accurate early diagnosis and prompt efficient treatment.

9. Bilateral exploratory burr holes should be made if there is a reasonable suspicion of the diagnosis. It is much better to have a negative exploration than an operation carried out too late or not at all.

10. Extradural hæmorrhage may occur over the cerebellum; or a large extradural hæmorrhage over the cerebral hemisphere may lift the lateral sinus out of its bed and extend back over the cerebellum.

11. Three pathological lesions may simulate the syndrome of extradural hæmorrhage: (*a*) Bruising and œdema of the brain; (*b*) Intracerebral hæmorrhage; (*c*) Acute subdural hæmorrhage.

In the last analysis exploratory burr holes establish or disprove the diagnosis.

REFERENCES

[1] JACOBSON, W. H. A., "Middle Meningeal Hæmorrhage", *Guy's Hosp. Rep.*, 1885–86, xliii, 147.

[2] KEITH, W. S., Personal communication.

SPINAL EPIDURAL SUPPURATION, WITH SPECIAL REFERENCE TO OSTEOMYELITIS OF THE VERTEBRÆ

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INTRODUCTION

Suppuration within the spinal epidural space is a dangerous condition which proves fatal unless diagnosed and treated surgically. It is an uncommon condition and therefore not often recognized, and the literature for the most part contains accounts of single cases only.[1, 2] Three authors,[3, 4, 5] however, have embodied in their reports an analysis of cases previously recorded, and deduce therefrom a syndrome very suggestive of the pathological process going on.

The present paper is based on a personal experience of 7 cases occurring during the last six years, and describes a sign that was constant in 5 of them.

ANATOMICAL CONSIDERATIONS

The spinal epidural space is the space lying between the spinal dura and the walls of the spinal canal. It extends from the foramen magnum above to the lower end of the sacral canal below. Except for that part distal to the second sacral vertebra, where it surrounds the termination of the dural envelope and the lowest spinal nerves, it does not extend on to the anterior or antero-lateral aspects of the cord at all, as the dura here is closely applied to the corresponding parts of the bony canal: it lies entirely on the posterior and postero-lateral surface of the dura. It is filled with fat and areolar tissue containing blood-vessels and lymphatics, and, as Pincoffs[5] has shown on the cadaver by injecting radiopaque fluid up the spinal canal through the sacral hiatus, it sends extensions into the intervertebral foramina along the nerve-roots. The fat varies inversely as the thickness of the cord, being greatest over the thoracic segments and least over the cervical and lumbar enlargements.

These anatomical considerations explain why suppuration occurs only on the dorsal aspect of the dura; how it sometimes passes outwards through the intervertebral foramina to involve the perinephric tissues, thoracic cavity, etc.; and why, since the dura is firmly attached to the margins of the foramen magnum, it never passes into the cranium.

NOMENCLATURE

This inflammatory process has been variously described as perimeningitis spinalis, peripachymeningitis spinalis, pachymeningitis spinalis externa, and epimeningitis spinalis; but since it is not primarily a meningitis at all, the terms spinal extradural, or epidural, suppuration are more accurate, and the latter will be the one used in this paper.

PATHOLOGY

Like suppuration elsewhere it may arise by direct infection, direct extension, or via the blood or lymphatic stream. The first of these is rare in civil practice, though bullet and shrapnel wounds must have accounted for cases during the War: the accompanying injury to the soft tissues, vertebræ, and spinal cord, however, would render them quite different from those under review now. The use of a dirty lumbar puncture needle might initiate it, and, as a corollary, one would think that a properly executed lumbar puncture in the presence of purulent meningitis might result in infection of the epidural space. Yet this rarely occurs and most authors deny its existence, though Peters[3] regards it as a definite cause. From the practical point of view direct infection of the space almost never occurs. Direct extension from contiguous structures is the commonest mode of infection: in the present series 3 cases were secondary to acute staphylococcal osteomyelitis of laminæ, and 1 case to tuberculous disease of a lamina; 1 case resulted from a suppurating tubulodermoid passing through a spina bifida; and 1 case from a suppurating sacrococcygeal sinus. The seventh case was hæmatogenous, and, like the tuberculous one, gave rise to a localized abscess surrounded by fibroblastic reaction in the epidural space. Dandy[4] thinks that some cases result from spread along adjacent lymphatics, and quotes two following carbuncle in the dorsal region in which the epidural suppuration developed at the same level.

Spinal epidural suppuration may be classified into two types: (1) The spreading type; and (2) The encapsulated type.

CASE REPORTS

Case 1.—My first case was referred to me by Professor K. D. Wilkinson in 1932.

HISTORY.—He was a healthy athletic youth of twenty years, well known in the Midlands as an amateur footballer. In August, 1932, he was badly sunburnt at Torquay and developed a mild skin infection which led to furunculosis. He returned home feeling out of sorts, but played football on Sept. 30. After the game he complained of intense pain in the right buttock and down the back of the right thigh, and when he reached home he stood by the fireplace, leaning on the mantelpiece, with the right leg and thigh flexed, and there he remained motionless for three hours, so agonising were the smallest movements. Eventually he was carried to bed and his doctor was called in. The temperature was 103° F., and he was transferred to a nursing home under the care of Professor Wilkinson. He was delirious, had a dry tongue, and became very constipated, and in addition to the pain in the right leg complained of increasing pain in the lumbar region. On Oct. 7 he developed stiffness of the neck and a bilateral Kernig's sign, and the temperature was 103° F. and pulse-rate 110. A lumbar puncture failed because of inability to flex the spine. R.B.C. 4,800,000; W.B.C. 28,000.

ON EXAMINATION.—I saw him on Oct. 9, and attempted to do a lumbar puncture, but could not get in as he would not permit the least flexion of the spine, though he did not object to hyperextension. A cistern puncture produced clear fluid under increased pressure. It contained 70 cells, and a pure culture of yellow staphylococci was grown. Because of this and the spinal pain and rigidity an osteomyelitis of a lumbar vertebra was suspected, but could not be located, and X rays failed to show any abnormality.

He continued in much the same way for another week, and then complained of pain in the right loin and developed some tenderness and rigidity there. A second X-ray examination again failed to reveal any bone lesion, but the line of the outer border of the psoas muscle was obliterated, and a perinephric abscess was diagnosed.

OPERATION.—

On Oct. 31 a fairly large perinephric abscess was opened and there was improvement for several days, but the high temperatures returned. He refused to attempt any

active movements of the legs, and passive ones were extremely painful. The reflexes were diminished and all sensations were impaired. There was no incontinence.

Early in December he developed some œdema over the 1st and 2nd lumbar spines.

On Dec. 3 an incision over and down to the 1st lumbar spinous process was made. Pus was found and the lamina on the right side was necrotic. The necrotic bone was removed and an abscess cavity inside the spinal canal was revealed and drained.

PROGRESS.—The progress from now on was satisfactory. The temperature and pulse subsided and the pain slowly diminished. He developed sacro-iliac arthritis on the right side, but this cleared up with radiant heat treatment. After small fragments of bone had discharged from the wound, it healed soundly by March, 1933.

During the autumn of 1933 he started playing football again and has been in perfect health ever since.

Comment.—Had this epidural abscess not succeeded in discharging into the perinephric tissues this patient would almost certainly have died.

Case 2.—The second case was a child of 14 years sent into the General Hospital, Birmingham, by Professor Wynn on July 22, 1934.

HISTORY.—On July 10 she had been running about in the heat and had bathed in the river, after which she felt out-of-sorts and complained of headache. She went home and lay down on the sofa. Next day she still felt poorly and lay on the sofa most of the time. On the following day she complained of slight aching in the lower part of the back. This rapidly increased and she found she could get more comfort by sitting erect in a chair than by lying down. Then she developed a temperature and the pain became so bad that morphia was necessary at night. By the eighth day of the illness she could not sit in a chair but preferred to stand up leaning against the head of the bed, and drugs were necessary to obtain any rest at all. On the eleventh day she suddenly lost the use of both legs. On the twelfth day a lumbar puncture was performed and pus was withdrawn, presumably from the epidural space, and considerable relief was given. This was the stage at which Professor Wynn was called in, and he transferred her to the General Hospital and asked me to see her.

ON EXAMINATION (July 22, 1934).—The child looked ill and was feverish. There was a flaccid paralysis of both legs. The left plantar was the only reflex present and this was flexor. There was loss of sensation to pain and touch below the level of the 11th dorsal segment. Retention of urine and constipation were present. There was rigidity of the spine including the neck, though hyperextension could be obtained. There was an area of œdema over the dorsolumbar region and marked tenderness over the 8th dorsal spine. The remainder of the central nervous system was normal except for the presence of nystagmus.

Having in mind *Case* 1, a diagnosis of osteomyelitis of the spine with epidural abscess and meningitis was made, and it was decided to attempt drainage.

OPERATION.—Laminectomy centred over the 10th dorsal vertebra was made. On separating the muscles from the laminæ pus under tension was encountered, and when bone was removed the epidural space was found to contain pus. The laminectomy was extended up to the 8th dorsal spinous process and down to the second lumbar, yet the whole of the exposed epidural space was filled with pus. It was decided that the child had had as much as she could stand in her toxic state, and small tubes were passed upwards and downwards into the epidural space, and the wound was packed with flavine packing. The 11th dorsal lamina was necrotic.

PROGRESS.—On July 23 her general condition was better. Nystagmus had disappeared, though neck rigidity was still present. Reflexes were returning. Incontinent.

On July 26 neck rigidity was still present. For this reason a cisternal puncture was performed and cerebrospinal fluid under pressure was obtained: its chemistry and cytology were normal and it was sterile.

Following this she improved slowly, running a pyrexial course for months, and she developed bed-sores. The back healed and the temperature had settled by December, 1934, but she remained paraplegic.

A letter from her doctor in December, 1937, states that though well and cheerful she has contractures of the legs and requires regular catheterization. No pain or headaches. Practically anæsthetic in the lower limbs and quite paralysed.

Comment.—I believe I ought to have extended the laminectomy until normal epidural fat had been reached, and should do so in a similar case. The operation certainly saved her life.

The following case, chronologically out of order, was another example of osteomyelitis. The child died and the notes of the autopsy are included.

Case 3.—A girl, aged 10 years, was admitted on Aug. 22, 1936, to the Birmingham Children's Hospital under the care of Dr. Smellie, having developed "influenza" seven days previously. She suffered headache and lassitude and two days previously had developed pain in the back and neck and become feverish. She was a well-developed child, but lay on her right side with the head retracted and in a position almost of opisthotonos. She was quite rational, but complained of considerable pain in the neck and back. The neck rigidity was extreme and there was Kernig's sign bilaterally. The cranial nerves were normal, but an early bilateral papillœdema was present. Muscle power and tone were fairly good, but the abdominal reflexes and knee-jerks were absent and the plantar reflexes indeterminate. A lumbar puncture was performed and 10 c.c. of thick purulent fluid trickled slowly from the needle. It contained innumerable pus cells and Gram-positive cocci in various formations. Next day the patient was in much the same condition. Lumbar puncture produced 7 c.c. of purulent fluid. The subsequent history was as follows:—

Aug. 24, 1936, condition unchanged. Lumbar puncture gave 2 c.c. of purulent fluid.

Aug. 25, less restless but otherwise not improved. Lumbar puncture—2 c.c. of thick greenish fluid with foul odour, followed by 25 c.c. of thin but turbid fluid. It was thought that the condition was one of meningococcal meningitis and serum was given.

Aug. 26, mildly delirious. Lumbar puncture at 10 a.m.—15 c.c. of opalescent fluid withdrawn. At 6 p.m. another lumbar puncture—15 c.c. of opalescent fluid containing 3200 cells per c.mm.

Aug. 27, pain in the back less severe. Quite rational. Lumbar puncture—20 c.c. turbid fluid containing 1610 cells per c. mm. Sugar 0 mg. Protein 30 mg. Chlorides 600 mg.

Aug. 28, complaining of pain in left loin. Examination reveals tenderness and fulness in the loin.

Aug. 29, developed severe headache. Lumbar puncture—10 c.c. fluid removed.

Aug. 30, headache better, but still has much neck and spine rigidity. X-ray examination of spine revealed no abnormality. Lumbar puncture produced 40 c.c. of foul-smelling thick pus.

It was at this stage that I was asked to see the child. I diagnosed a perinephric abscess on the left side, and having in mind the last two cases, felt that the full sequence of events was osteomyelitis of one of the lumbar vertebræ giving rise to spreading suppuration within the epidural space with secondary ascending spinal meningitis, and spread, by continuity, through an intervertebral foramen, into the perinephric tissues. I was going away next day for my holiday so asked that a colleague should take on the case.

Aug. 31, perinephric abscess opened and drained.

Sept. 1, patient comfortable, but neck rigidity more marked. Lumbar puncture—12 c.c. of thin pus removed.

Sept. 2, no change. Lumbar puncture—15 c.c. of thin pus removed.

Sept. 3, fatal reactionary hæmorrhage.

POST-MORTEM EXAMINATION.—Osteomyelitis of body of 1st lumbar vertebra. Pus in the epidural space. Spinal meningitis. Basal meningitis. Psoas and perinephric abscess.

The next two cases gave a less dramatic picture, for the illness in each instance was essentially subacute rather than acute and fulminating as in the three already described.

Case 4.—A child of 2 years was admitted to the Birmingham Children's Hospital on Nov. 11, 1936, under the care of Professor L. G. Parsons. Until six weeks previously this little boy had been quite well. He then became tired and irritable and three weeks ago was noticed to walk with a rather awkward gait. During the week before admission to hospital he showed a reluctance to bend his back, and would not stoop without holding on to something.

ON ADMISSION.—He was a sturdy, well-nourished but peevish child. The lumbar spine was held stiffly and was tender to pressure. The central nervous system, except for a diminished knee-jerk on the left, was normal. The condition was thought to be a spinal caries, though the X-ray picture revealed nothing abnormal.

On Nov. 17 Mr. G. P. Mills was asked to see him, and he pointed out that although the child would allow no flexion of the lumbar spine hyperextension caused no discomfort, and for this reason he suggested some form of chronic spinal meningitis. It is noteworthy that at that time the lumbar spine only was affected.

Nov. 19, lumbar puncture—slightly turbid fluid under a pressure of 300 mm. was withdrawn. It contained 462 cells per c.mm. and hæmolytic *Sta. albus* was cultured from it.

ON EXAMINATION.—I was asked to see the boy on Dec. 1. The child was in remarkably good condition but still held the lumbar spine stiff, and was uncomfortable and irritable. On examining the lumbosacral region one found a tiny sinus to one side of the midline of the upper part of the sacrum. Running upwards from this to the lumbosacral junction a fibrous cord could be felt beneath the skin. An X-ray picture of this region revealed a spina bifida of the 5th lumbar vertebra; and the whole picture suggested a tubulodermoid communicating with the spinal canal. It was only one more step to suggest that mild infection had spread along this tract and involved the epidural space, thereby causing a low-grade ascending spinal meningitis. I advised surgical exploration and drainage, but it was thought wiser to watch events for a few more days. However, as no improvement took place, surgical exploration was thought justifiable.

OPERATION.—On Dec. 5, laminectomy of the 4th and 5th lumbar and 1st sacral vertebræ was performed. The sinus was found to pass down to the region of the bifid 5th lumbar spine, at which point it widened out rapidly, becoming an encapsulated tumour inside the spinal canal, and extending upwards within the cone or the cauda equina. On incision, it proved to be a dermoid containing hair and sebaceous material. The contents were very gently scraped out, but it was thought dangerous to attempt removing the capsule for fear of spreading infection. The wound was closed by stitches and healed by first intention.

PROGRESS.—The spinal rigidity subsided and the child rapidly returned to normal.

The following case is of the same kind.

Case 5.—A boy of 4 years was admitted to the Children's Hospital, Birmingham, on April 18, 1937, under the care of Professor L. G. Parsons. Two weeks previously he had developed headache, vomiting, and pyrexia. Soon afterwards he complained of pain in the back, and began to lose weight. His parents had noticed that from birth he had had a small sinus at the bottom of the spine, and this sinus had begun to discharge four days before coming to hospital.

ON ADMISSION.—The temperature was 102·4° F. and the pulse 124. The facial expression was that of a perfectly normal healthy child, and mentally he was alert, but on turning down the bedclothes one found the knees drawn up, the spine arched backwards, and the head retracted, there being neck rigidity. The left pupil was larger than the right, but the cranial nerves were otherwise normal. The arms were rather spastic, but the reflexes were normal. The knee-jerks were absent, there was a bilateral Kernig's sign, and both plantar reflexes were extensor. Any attempt to straighten the spine was resisted, but further extension was painless. There was a small sacrococcygeal sinus discharging thin pus, and the surrounding skin was reddened. A lumbar puncture produced a few drops of thick ropy pus.

On April 19, lumbar puncture—3 c.c. of thick pus dripped out. It contained polymorphonuclear leucocytes and cultures yielded coliform bacilli and enterococci. A smear from the sacrococcygeal sinus showed Gram-positive cocci and a few *B. coli*. A few days later *B. proteus* was obtained.

April 21, cistern puncture—slightly turbid fluid containing 70 cells per c.mm. Fluid sterile.

April 22, general condition good. Neck rigidity had disappeared, though the spine was still markedly arched. Very irritable. Cistern puncture repeated.

April 23, lumbar puncture—several cubic centimetres thick pus obtained, which yielded *B. proteus* on culture.

From now on there was steady improvement, the pupils became equal, the plantar reflexes became flexor, and the neck rigidity disappeared. The spine, however, remained

rigidly hyperextended, and any attempt to flex it caused great pain, but further hyperextension could be obtained without discomfort. There was incontinence of urine and sometimes of fæces. The temperature remained raised. The sacrococcygeal sinus discharged freely.

During the next few weeks he varied, sometimes developing more stiffness of the spine, sometimes so much less that he could sit up, but in July he became uncomfortable and I was asked to see him. With the above history and with the experience of the previous cases, I diagnosed epidural abscess associated with spina bifida of the sacrum, and operation was advised.

OPERATION (July 10).—The sacral canal was explored by laminectomy. It contained thick pus, and the sacrococcygeal sinus was attached to the dura mater of the cord. The space was drained and the child made an uninterrupted recovery, the spinal movements returning to normal.

Cases 6 and 7 are instances of localized epidural abscess mimicking spinal cord tumour.

Case 6.—A youth of eighteen years, a slaughterer by trade, was admitted to the General Hospital, Birmingham, on May 29, 1934, with spastic paresis of both legs.

In 1930 he developed a septic thumb, and four months later furunculosis. One month after this he developed sudden severe pain in the lower part of the back, so severe that he could not sit up. He was admitted to Dudley Road Hospital, where he remained for six weeks, by which time the pain had disappeared. He remained well for a year and then had slight pain in the same region for four weeks. It again cleared up, and he remained well until October, 1933, when he experienced cramp-like pains in the right calf. Gradually the right leg became weak, and on three occasions it gave way and he fell to the ground. Early in May, 1934, he developed similar pains and weakness in the left leg, and found that when bending forwards to wash his face he became giddy if he closed his eyes. Latterly he had had incontinence of urine. He had also noticed that the right leg shook when it touched the ground. At the time of admission he could only just manage to stand without assistance. He had had no previous illnesses. His mother was in a sanatorium.

ON EXAMINATION.—Everything above the level of the umbilicus was neurologically normal. Both legs were extremely spastic. The left one could just be raised from the bed, but not so the right. Much exaggerated knee-jerks, bilateral knee and ankle clonus, and bilateral Babinski sign. Impairment of all sensations below the level of the 11th dorsal segment. Lumbar puncture revealed a fairly complete spinal block, and the cerebrospinal fluid was clear, but yellow. Sugar 36 mg., NaCl 711 mg., and protein 360 mg. per 100 c.c. There were 18 cells per c.mm. Wassermann reaction negative. Radiographs of the spine were normal. A diagnosis of spinal-cord tumour was made, but confirmation of the exact level was sought by injecting 2 c.c. of lipiodol into the cisterna magna: it was held up at the 9th dorsal vertebra.

OPERATION.—On May 30, laminectomy centred over the 8th dorsal spinous process. The extradural fat for about two inches was replaced by dense fibrous tissue, firmly adherent to the dura. It was not adherent to the periosteum. Its nature was not appreciated: indeed I thought it was a fibroma or meningioma, and felt that the best way to find its limits was to open the dura where it looked normal and to work from this point. This was done, but the mass broke during dissection and two minute abscesses were revealed. The mass at its thickest measured half an inch. Wound clean stitched.

PROGRESS.—The convalescence was very stormy. He ran high temperatures and suffered extremely severe posterior root pain with crushing girdle pains. He showed signs of an ascending spinal meningitis and the wound became septic. However, after four perilous days the condition settled, and he was discharged healed on June 30. He returned to his work as a slaughterman in September, 1934, and has remained quite well ever since.

Comment.—On reviewing the full story there can be little doubt that the original infection occurred when he developed the sudden pain in the back in 1930, one month after the furunculosis.

Case 7.—On July 2, 1934, a miner, aged 54 years, was admitted to the General Hospital, Birmingham, complaining of interscapular pain of eight or nine years' standing.

It was aggravated by work and he also had had pain in the lumbar region. Thirteen weeks previously he had developed pain in the left side of the chest from the 8th to the 12th ribs. It extended down into the abdomen and soon affected the right side of the chest and abdomen too. Six weeks before admission he had experienced numbness and weakness in the left leg, and walking became difficult; three weeks later the right leg became similarly affected, and for the past two weeks he had been bed-ridden. Latterly, painful involuntary contractions of the legs had occurred.

Until thirteen weeks previously he had been a strong healthy athletic man and had suffered no illnesses. He had, however, been losing a little weight. He had two healthy children.

ON EXAMINATION.—Cranial nerves normal. Upper limbs normal. Lower limbs in a state of spastic paralysis. Impairment of pain and thermal sensations up to level of umbilicus: tactile sense unimpaired, however. Abdominal reflexes absent, knee-jerks exaggerated, ankle-jerks exaggerated and clonus present, plantar reflexes extensor. Radiographs of the dorsal spine showed osteo-arthritis. Lumbar puncture showed evidence of spinal block, and the cerebrospinal fluid contained 800 mg. of protein per 100 c.c. A diagnosis of spinal cord tumour at the level of about the 5th dorsal spinous process was made.

OPERATION (July 14).—Laminectomy centred over the 5th dorsal spinous process, and involving the 3rd–6th dorsal laminæ. The laminæ of the 5th vertebra were very brittle and dry, and the epidural fat was replaced by encysted gelatinous granulation material producing pressure on the cord. It was gently mopped out and the wound was closed in the usual way. Microscopic examination showed this material to be tuberculous.

PROGRESS.—The wound healed satisfactorily, and a month later he was sent in a plaster bed to the convalescent annexe. Two months later, however, the wound broke down and he developed signs and symptoms in the lungs. These became rapidly worse, and he died on Nov. 28.

POST-MORTEM EXAMINATION.—Autopsy revealed that there was tuberculous disease of the bodies of the 4th–8th dorsal vertebræ, though no collapse. There was extensive disease in the right lung and marked involvement of the tracheo-bronchial glands.

DISCUSSION AND CONCLUSIONS

In reviewing these 7 cases of spinal epidural suppuration one may at once divide them into: (1) diffuse, and (2) localized; and the former may be subdivided into (*a*) acute fulminating, and (*b*) subacute. The two instances of localized suppuration produced their dramatic symptoms by mechanical pressure on the cord and were, indeed, tumiform lesions giving signs indistinguishable from those of neoplasm within the spinal canal—namely, pain, sensory and motor changes, and alteration in the manometric pressure readings and the chemistry of the cerebrospinal fluid. In the 5 cases of diffuse suppuration there were certain features common to all. All complained of pain in the back as the first symptom; all developed stiff spines which on analysis proved to be a matter of absolute limitation of flexion whereas hyperextension was still free and painless, a point of distinction from tuberculous disease of the spine; in all cases this stiffness slowly or rapidly came to involve more and more of the spine, until, in some, neck rigidity and even head-retraction developed, this march of events giving the impression of an inflammatory process steadily rising up the spinal canal, or of an ascending spinal meningitis secondary to it. It is probable that both events take place side by side, and certainly in three instances the cerebrospinal fluid from cistern puncture showed cell increase or frank infection. Another common feature was pyrexia; and I believe that the combination of pain in the back, limitation of flexion but free hyperextension of the spine tending to involve higher and higher levels in a young patient, with a temperature, means ascending spinal meningitis secondary to infection

within the spinal canal but outside the dura. Osteomyelitis is undoubtedly the commonest cause, yet its diagnosis in the spine is peculiarly difficult, and most clinicians have been humbled by it. The three cases in this series were typical of the condition in that they were all under the age of 20 years, all began with suddenness, all presented very ill people, yet all defied diagnosis for many days. In retrospect one feels that more effort should have been made to find local tenderness. It is obvious that, with such a combination of circumstances, lumbar puncture carries the gravest risks, though useful information might be obtained by inserting the needle into the epidural space only. One case presented slight œdema over the dorsolumbar spine, and this, on a future occasion, would suggest deep inflammation and put one on guard.

It appears to me, therefore, that several conclusions may be drawn from a study of these cases: firstly, that there is a combination of signs and symptoms indicating ascending spinal meningitis secondary to infection in the spinal epidural space; secondly, that if the patient is not very toxic some such insignificant abnormality as a tubulodermoid or sacrococcygeal sinus must be sought for, or a septic embolism in the epidural fat not yet shut off into an abscess, be considered; thirdly, that if the patient is very ill, with high temperature and great pain, and, if in addition, there is a tender zone in the spine or slight œdema, the cause is osteomyelitis of a vertebra; and finally, in the latter case immediate surgical intervention is urgent. This is so not only to relieve toxæmia, but because paraplegia may occur at any moment, and when it does occur it develops so rapidly—within twenty-four hours—and clears up so slowly or not at all in spite of drainage of the abscess that its genesis must be vascular.

The course of inflammation may be resolution, suppuration with tissue death, or healing with fibrosis, and it is interesting to speculate as to the effect of an inflammatory process in the epidural fat healing by fibrosis. Fibrosed fat must be much less yielding during spinal movements than normal fat, and pull on the sheaths of the nerve-roots must occur and account for limitation of full movements. Some cases of stiff back and of atypical sciaticas in the absence of osteoarthritis must be due to such fibrosis, and the efficacy of epidural injection in sciatica associated with scoliosis, or spinal manipulations in painful back, must be due to the breaking down of such adhesions. In trauma, too, epidural hæmorrhage would be absorbed and replaced by fibrous tissue.

Since writing the above an eighth case has come into my hands:—

Case 8.—A youth of 15 years, who two weeks before had begun to get pains in the lumbar region. A week before he had fallen off his bicycle, but so far as he could recall had not injured himself. During the four days prior to admission he had felt shivery and had sweated a good deal, and thirty-six hours before admission had felt weak and numb in the legs. This rapidly developed into a complete paraplegia and he had retention of urine.

On Admission (April 23, 1938).—When admitted to the General Hospital, Birmingham, he was completely anæsthetic below the 11th dorsal segment, there was a flaccid paralysis of both legs, and the only reflexes present were the plantars, which were flexor. He had great pain in the lumbar region and acute tenderness over the 1st lumbar spinous process, with œdema to the left side of it. The spine was held stiff, so that no flexion could be obtained, though a little hyperextension was possible, and there was neck rigidity. The pulse was 110, but the temperature was only 98° F. Nevertheless, there could be no doubt as to the diagnosis, and operation within two hours of coming in was performed.

Operation.—A central incision over the 12th dorsal and 1st and 2nd lumbar spines was made. Pus under considerable tension welled up from the left side of the 1st lumbar

region, and the lamina on the left side was eroded and the spinal epidural space open. Necrotic bone was nibbled away, and it was necessary to remove the laminæ of all three vertebræ before healthy epidural fat was reached. In this case the pus appeared to be between the fat and the dura and oozed out from several holes in the fat. The dura itself, however, was healthy. The wound was drained and the patient returned to bed.

PROGRESS TO DATE.—After two days the plantar reflexes had disappeared, but after a further two days returned but had become extensor. At the present time (end of August) there has been a little return of sensation but no recovery of muscle power, and he still requires catheterization.

BIBLIOGRAPHY

[1] ABRAMSON, MCCONNELL, and WILSON, " Acute Epidural Spinal Abscess ", *Brit. Med. Jour.*, 1934, i, 1114.
[2] MINTZMAN, J., " A Case of Epidural Spinal Abscess ", *Ibid.*, ii, 593.
[3] PETERS, R., " Ueber die Entzündung der extraduralen Gewebes des Rückemarks bei der Gewickstarre ", *Deut. med. Woch.*, 1906, xxxii, 1151.
[4] DANDY, W. E., " Abscesses and Inflammatory Tumours of the Spinal Epidural Space ", *Arch. of Surg.*, 1926, xiii, 477.
[5] PINCOFFS, M. C., " Purulent Spinal Perimeningitis ", *Trans. Assoc. Amer. Phys.*, 1926, xli, 247.

RECURRENT DISLOCATION OF THE SHOULDER
A PLEA FOR A SIMPLE OPERATION

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During recent years in looking over current surgical literature I have been much surprised at the number and complexity of the operations proposed for recurrent dislocation of the shoulder. In the present year there have been three articles: one by Boicev[1], in which no less than twenty methods are briefly described and reference is given to some eighty-seven papers on the subject; the second by Horwitz and Davidson,[2] who have published a critical appreciation of Nicola's method; and the third, in the last number of this Journal, by Blundell-Bankart,[3] who has given an excellently illustrated account of his method, which bears exceedingly close resemblance to that described by Putti and various French and Scandinavian authors.

I think everyone will admit that it was Clairmont's[4] operation of 1909 which gave a great impetus to this subject. He utilized the anterior portion of the deltoid

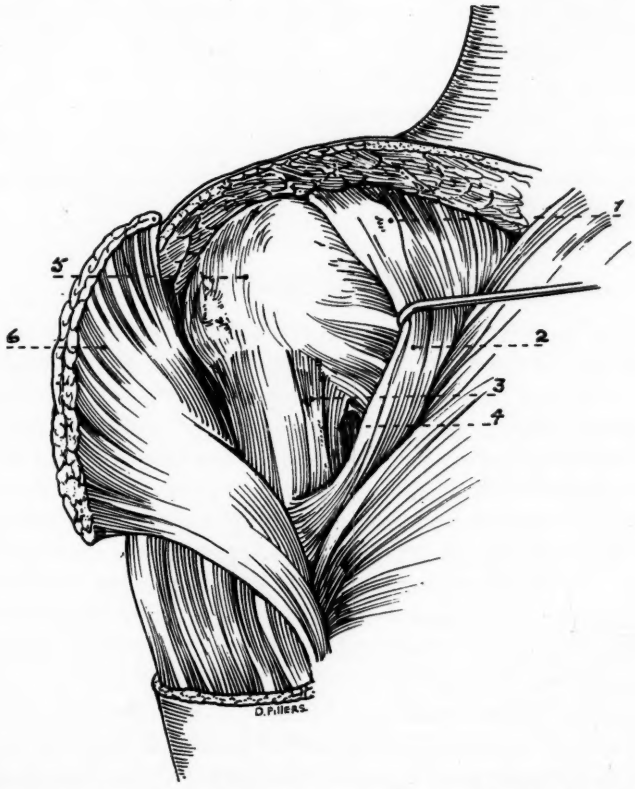

Fig. 259.—Dissection showing the anterior aspect of the shoulder-joint. 1, Coracoid process; 2, Coracobrachialis; 3, Long head of the biceps; 4, Quadrilateral space, through which the graft is put; 5, Head of the humerus in the capsule; 6, Anterior part of the deltoid, cut and turned back.

muscle, which was pushed through the quadrilateral space under the capsule of the shoulder-joint and then fixed in position through a posterior incision. This operation had, however, several drawbacks: it sacrificed part of the most important shoulder muscle; a muscular band is a poor substitute for a ligament; the posterior attachment of the muscle found no fixed bone support; and, lastly, two considerable incisions were necessary to carry out the procedure.

It is not surprising, therefore, that the Clairmont operation only gave a certain proportion of successes and that further attempts were made to improve upon it. It is not my purpose here to review or discuss these various proposals. Such reviews have been recently undertaken by Boicev,[1] Speed,[5] Henderson,[6] and Willard.[7] The procedures may be divided into two main categories—namely, utilization of the long tendon of the biceps or other tendon passed through the head of the humerus, anchoring it to the coracoid, and exposure of the anterior portion of the capsule as described by Bankart and Putti, with various methods of capsulorrhaphy (suturing the glenoid ligament, building up a new glenoid ridge, or adding a tibial graft).

I am very much impressed with the care and thought which have been given to these methods, and I do not doubt that their use in skilled hands will give a very high proportion of success. But I cannot believe that such highly complicated methods are really necessary. They all involve large incisions, free opening of the joint, and sacrifice, or at any rate injure, some of the important muscles around the shoulder.

Since 1923 I have been using a very much simpler method, which was really an outcome of the Clairmont operation. But a description of this method has only appeared in general articles on fascial surgery,[8, 9, 10] and for this reason it has found no place in text-books or reviews. I have, however, carried it out in 10 cases with uniformly successful results, and I feel therefore justified in putting in a plea that its use might be considered in preference to the much more difficult operations which are in current vogue.

THE FASCIAL SLING OPERATION

The principle is that of Clairmont—that is, to provide a sling around the damaged capsule of the joint—but to do this by using a free graft of fascia lata instead of a slip of deltoid muscle. An incision $\frac{3}{4}$ in. long is made over the anterior border of the deltoid muscle about 1 in. below the coracoid process. The interval between the deltoid and the pectoralis major having been defined and opened, the tip of the index finger is inserted into this, keeping close to the capsule of the shoulder-joint. No difficulty is experienced in pushing the finger through the quadrilateral space, until it reaches the back of the joint, where it can be felt deep to the skin and muscles. This tunnel below the joint having been formed by the finger, a long pair of artery forceps is passed into it and made to project behind the posterior border of the deltoid muscle. A second incision is made about $\frac{3}{4}$ in. long over the point of the forceps, so that the latter can project on to the surface. There now exists a tunnel right underneath the capsule of the shoulder-joint. From the incisions at each end of this tunnel a large artery forceps is passed deep to the deltoid muscle upwards to the middle of the acromion process. A third incision $\frac{3}{4}$ in. long is made over this process of bone and the points of the artery forceps are made to emerge upwards through this incision. A piece of ribbon gauze is then threaded

through from the superior incision down to the anterior, then to the posterior, and back to the superior. This gauze is used to give the length of the fascial graft required. A piece of fascia of this length about 1½ in. wide is cut from the thigh, and is then threaded through the three tunnels in front, below, and behind the shoulder-joint, with the two free ends projecting above the acromion. These two ends are then sewn together with stout catgut and the three incisions are also sutured. The arm is held by an ordinary sling and can be used freely for under-arm movements after the stitches have been removed on the tenth day. At the end of a month over-arm movements can be undertaken. (*Figs.* 259–261.)

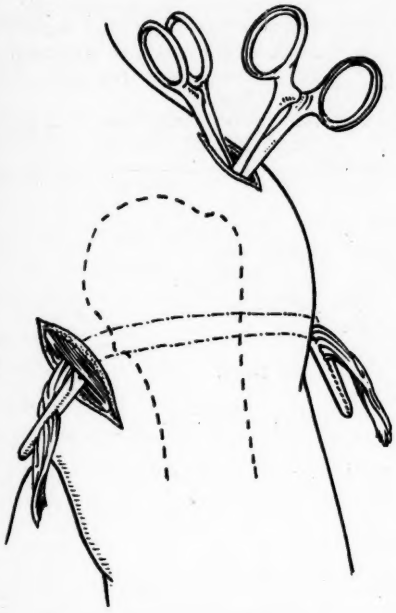

FIG. 260.—Diagram showing the fascial sling below the shoulder-joint, with two pairs of forceps ready to pull the ends of the sling upwards.

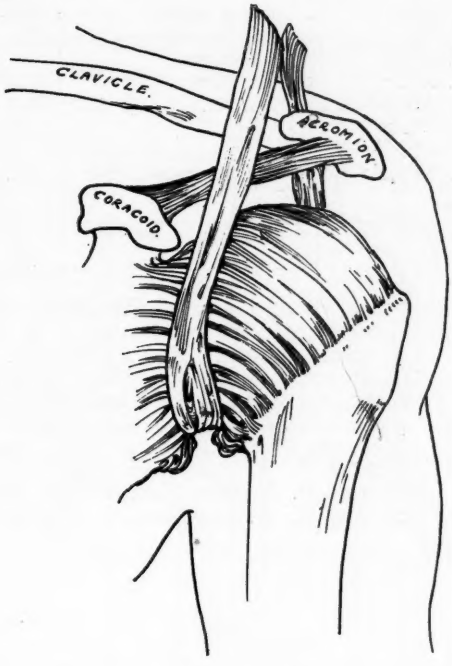

FIG. 261.—Diagram showing the fascial sling in position.

My series of cases has, of course, only been small, but success has been uniform and the course of recovery quite smooth. Some of the patients have been engaged in the most laborious occupations; others have been young and addicted to sport. The operation is one of great simplicity, it involves no hæmorrhage, exposure of the joint, or injury to muscles, and including the excision of the piece of fascia, only occupies about half an hour.

The Table gives details of the 10 cases in which this method has been employed. *Case* 6 is the only not *completely satisfactory* result in the series. All the cases up to end of 1931 were examined in 1931, and all were absolutely satisfactory (including *Case* 1) except *Case* 6, who was a rather feeble-minded epileptic. He came with a relative and said he had dislocated the arm once since operation, but he also said he could dislocate the other arm. He was very anxious to come in again for further operation. I remember the discussion at the time with the relative, and that it

was quite uncertain if he had dislocated the arm since. It was decided in any case that he was unsuitable for further treatment.

CONSECUTIVE LIST OF ALL CASES OF RECURRENT DISLOCATION OF THE SHOULDER IN WHICH THE OPERATION WAS USED

SEX	AGE	OCCUPATION	OPERATION DATE	RESULT
1. Male	40	Blacksmith's striker	Sept. 5, 1923	Back at full work in 3 months
2. Male	25	Labourer	Mar. 3, 1924	Back at work in 6 months
3. Male	34	Labourer	May 5, 1924	Back at work in 6 months
4. Male	37	Cigar maker	May 2, 1925	Back at work in 2 months
5. Male	25	Saddle maker	Nov. 4, 1927	Back at work in 2 months
6. Male	27	Labourer	Oct. 6, 1928	Satisfactory, but an epileptic
7. Male	31	Farmer	Aug. 18, 1931	Back at full work in 1 month
8. Male	21	Farmer	Oct. 22, 1931	Back at full work in 1 month
9. Male	17	Clerk	Dec. 18, 1937	All movements free and normal in 6 weeks
10. Female	25	House duties, golf	Jan. 19, 1938	All movements free and normal in 6 weeks

REFERENCES

[1] BOICEV, BOICIO, *Chir. d. Org. di Movimento*, 1938, March, 354.
[2] HORWITZ, M. T., and DAVIDSON, A. J., *Surgery*, 1938, July, 71.
[3] BLUNDELL-BANKART, A. S., *Brit. Jour. Surg.*, 1938, July, 23.
[4] CLAIRMONT, *Arch. f. klin. Chir.*, 1909, lxxxix, 798.
[5] SPEED, K., *Surg. Gynecol. and Obst.*, 1927, xliv., 468.
[6] HENDERSON, M. S., *Jour. Amer. Med. Assoc.*, 1930, xcv, 1653.
[7] WILLARD, DeF. P., *Ann. of Surg.*, 1936, ciii, 438.
[8] GROVES, E. W. HEY, *Bristol Med.-Chir. Journal*, 1933, 1, No. 187.
[9] GROVES, E. W. HEY, *Ann. of Surg.*, 1934, c, July, 20.
[10] GROVES, E. W. HEY, *Medical Annual*, 1925, 231.

GRANULOMATOUS ULCERS RESEMBLING TUBERCULOUS ULCERS OF THE STOMACH

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Incidence of Tuberculosis of the Stomach.—Tuberculous ulcers of the stomach as compared with tuberculosis of other organs of the body are rare. This is illustrated in the Table below collected from the reports of various authors.

INCIDENCE OF TUBERCULOSIS OF THE STOMACH

AUTHOR	TOTAL NECROPSIES	NECROPSIES IN TUBERCULOUS SUBJECTS	CASES OF TUBERCULOSIS OF STOMACH	PERCENTAGE OF ALL CASES	PERCENTAGE OF TUBERCULOUS CASES
1. Clayton and Wilkinson	7,169	—	50	0·7	—
2. Eisenhardt	—	6,000	1	—	0·016
3. Glaubitt	12,528	2,237	47	0·37	2.10
4. Gossmann	5,800	2,360	18	0·30	0·80
5. Grusdew	18,593	—	23	0·12	—
6. Houbschmann	10,000	—	10	0·10	—
7. Hundrat	—	418	2	—	0·48
8. Letulle	—	180	1	—	0·55
9. Melchior	—	848	6	—	0·70
10. Ophuls	3,000	632	0	0	0
11. Orsos	6,280	—	5	0·80	—
12. Plamoeck	—	490	3	—	0·60
13. Robertson	8,401	—	0	0	—
14. Simmonds	—	2,000	8	—	0·4
15. Leeds Hospital	10,000	—	3	0·03	—
16. K.G. Hospital, Vizagapatam (1930–35)	446	55	0	0	0

Clinically also, tuberculous infection of the stomach is rare. According to Demol (1923), of 1568 operated cases of gastric ulcer only 3 (0·19 per cent) were tuberculous. Of 2500 gastric operations at the Mayo Clinic during 1912–15 only 1 case was that of tuberculosis of the stomach (Broders). Of 548 operated cases of gastric and duodenal ulcers in King George Hospital, only 3 (0·55 per cent) were granulomatous ulcers resembling tuberculous ulcers (1931–35).

Varieties of Tuberculous Infection of the Stomach Wall.—These may be of two kinds: (1) Primary tuberculous infection of the stomach wall; (2) Tuberculosis of the stomach secondary to tuberculosis elsewhere. The former is important owing to its rarity; and the latter, though not so rare, is important because of the difficulty of obtaining a successful treatment of systemic tuberculosis.

Sources of Infection.—These are various:—

a. Through the spleen (Reinhold).

b. Through the transverse colon (Reinhold).

c. Through the neighbouring lymph-glands (Beneke).

d. From the duodenum (Chiari).

e. Retrograde transportation from the lymph-glands into the gastric walls.

Diagnosis.—When extensive shallow ulcers with undermined edges occur in the pyloric region it becomes very difficult clinically to differentiate them from malignant disease of the pyloric antrum. After gastrectomy one of the three ulcers in our series, which appeared to the naked eye to be typically tuberculous, failed to show on histological examination any evidence of tubercle (*Case* 3). *Case* 1 was diagnosed as a case of malignant disease and a gastrectomy was performed. It was only after gastrectomy that the ulcer was found to be granulomatous, resembling a tuberculous ulcer, which on histological examination it was suspected to be. In *Case* 2 no gastrectomy could be performed as the general condition of the patient was very poor. While performing gastro-enterostomy, a good view of the ulcer could be obtained through the stoma. It resembled the ulcer described in *Case* 1. The gland removed for biopsy was suspected to be tuberculous on histological examination. *Case* 3 resembled the first in all its aspects, but on microscopical examination there was no evidence of tubercle in the sections examined. A gastrectomy was performed in this case.

CASE REPORTS

Case 1.—

HISTORY.—A Hindu male, aged 29, was admitted in 1933 for pain in the umbilical region of 1½ years' duration. Pain usually started three to four hours after a meal, and was relieved by involuntary vomiting. The vomit was copious, and contained no bile or blood, but partly-digested food.

ON ADMISSION.—The patient was found to be emaciated and anæmic. The tongue was coated, the teeth dirty, and pyorrhœa alveolaris was present. There was no visible tumour

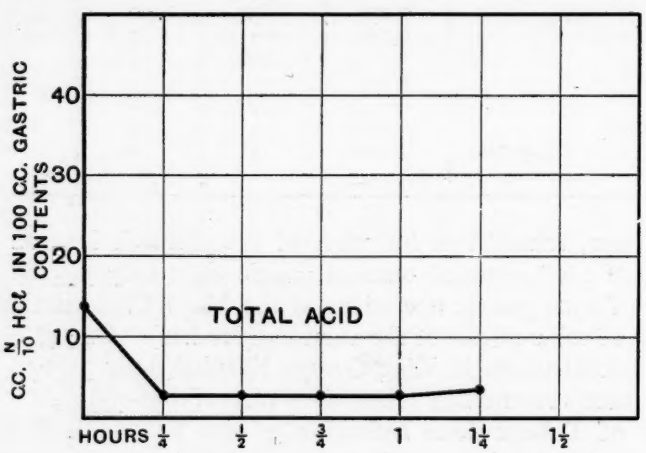

FIG. 262.—*Case* 1. Chart of test-meal examination, showing condition of achylia.

or peristalsis. There was tenderness on deep pressure at the umbilicus. No fluid was in the abdomen. There were no dilated veins. There were no masses to be felt anywhere in the abdomen. Spleen and liver were normal.

The respiratory and circulatory systems were normal. His blood-pressure was 90/60 mm. Wassermann reaction was negative. Urine was normal. Test-meal showed achylia (*Fig.* 262). Barium-meal examination showed a definite filling defect in the pars pylorica in all the

pictures of the stomach (*Fig.* 263, 1–4); the duodenal cap was not visualized. Radiographer suspected a neoplasm in the pars pylorica.

OPERATION.—He was operated upon under local anæsthesia, and on opening the abdomen a growth was felt in the pyloric portion of the stomach and the sub-pyloric group of glands

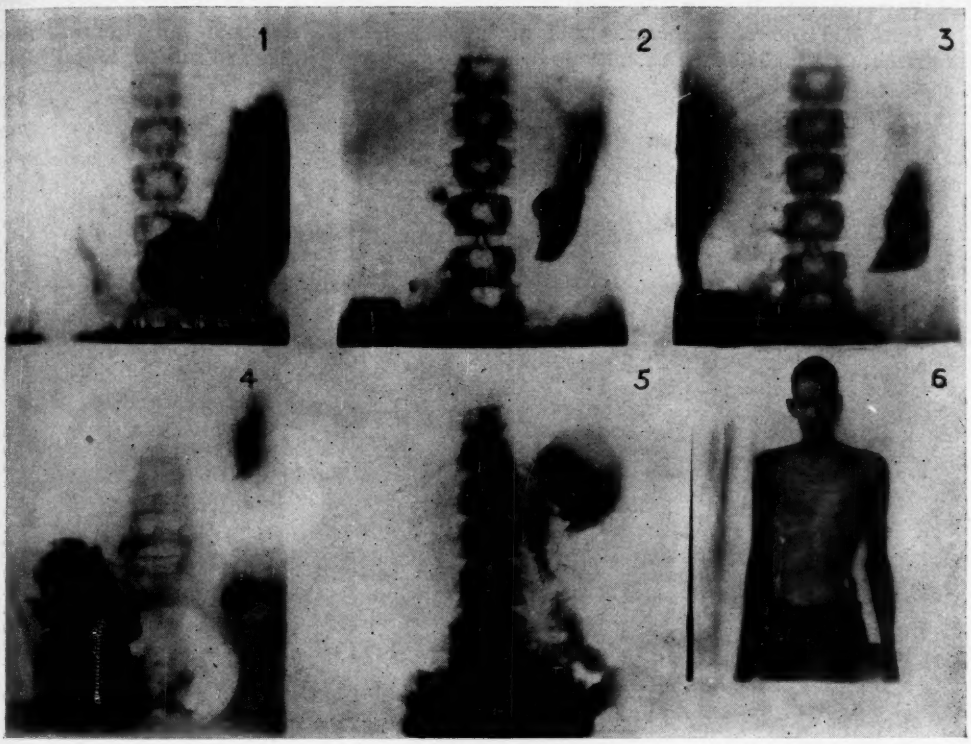

FIG. 263.—*Case* 1. 1—4, Radiographs of barium-meal examination, showing filling defects in stomach. 5, Barium-meal radiograph taken after partial gastrectomy and gastro-enterostomy (Balfour's method). 6, Photograph of patient before discharge from hospital.

FIG. 264.—*Case* 1. A drawing of the specimen.

was found to be enlarged. A partial gastrectomy with removal of enlarged glands was performed, Balfour's method being employed. A considerable number of enlarged glands were found along the superior mesenteric vessels, and in one portion definite enlargement of the lymph-channels were visible to the naked eye. The patient made an uneventful recovery and was treated subsequently by ultra-violet exposures to the abdomen (*Figs.* 263, 264).

PATHOLOGICAL REPORT (*Fig.* 265).—

Section A showed adenomatosis of the mucosa of the duodenum and granulomatous infiltration principally of the submucous coat involving the muscular and sub-peritoneal coats, suggesting tuberculosis.

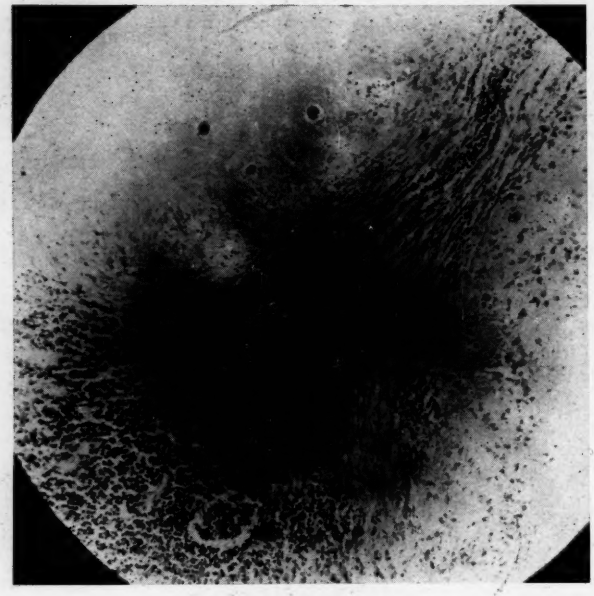

FIG. 265.—*Case* 1. A microphotograph of a section of the specimen.

Section B: Shallow chronic tuberculous ulcer; slightly undermined edges; the base of the ulcer is granulomatous; there are sub-peritoneal tubercles.

Section C, of the adjoining lymphatic gland, shows a tuberculous condition.

Section D, of another gland, shows reticulo-endothelial hyperplasia, both in the lymph-follicles and the pulp.

SUBSEQUENT HISTORY.—The patient has reported after the operation. He is keeping in good health. He was advised to take diluted hydrochloric acid and hepatex.

Case 2.—

HISTORY.—A Hindu female, aged 30 years, was admitted in 1934 for pain in the epigastrium of three months' duration. The pain and train of symptoms described below followed an attack of continuous fever for a month. She had no appetite for food and was severely constipated; she had severe pain in the epigastrium a quarter to half an hour after taking any food, liquid or solid—this was relieved by involuntary vomiting, which stopped the pain for a short time.

ON EXAMINATION.—There was a fullness in the epigastrium, with visible peristalsis from left to right. Blood examination showed slight anæmia. Urine was normal. Test-meal examination could not be done as the patient could not swallow the Ryle's tube, even after repeated attempts; her condition being unsatisfactory, this was not urged further. Barium-meal examination showed a filling defect in the pyloric region similar to that in *Case* 1 and the case was suspected to be one of granulomatous ulcer of the stomach (*Fig.* 266, 1–4). Her blood-pressure was 86/60 mm.

OPERATION.—She was operated upon under local anæsthesia. On opening the abdomen a thickening in the pyloric antrum was found, with enlarged sub-pyloric and coronary groups of glands and with two distinctly enlarged glands in the gastrocolic omentum. All the glands in front of the aorta were found to be enlarged. Some of the sub-pyloric group of glands were taken for biopsy. Gastrectomy was considered, but the condition of the patient being very poor a posterior no-loop gastrojejunostomy was done as a palliative measure. The ulcer was explored through the gastrojejunostomy opening before closing the stoma, and it resembled the ulcer of *Case* 1. The patient made an uneventful recovery. (*Fig.* 266.)

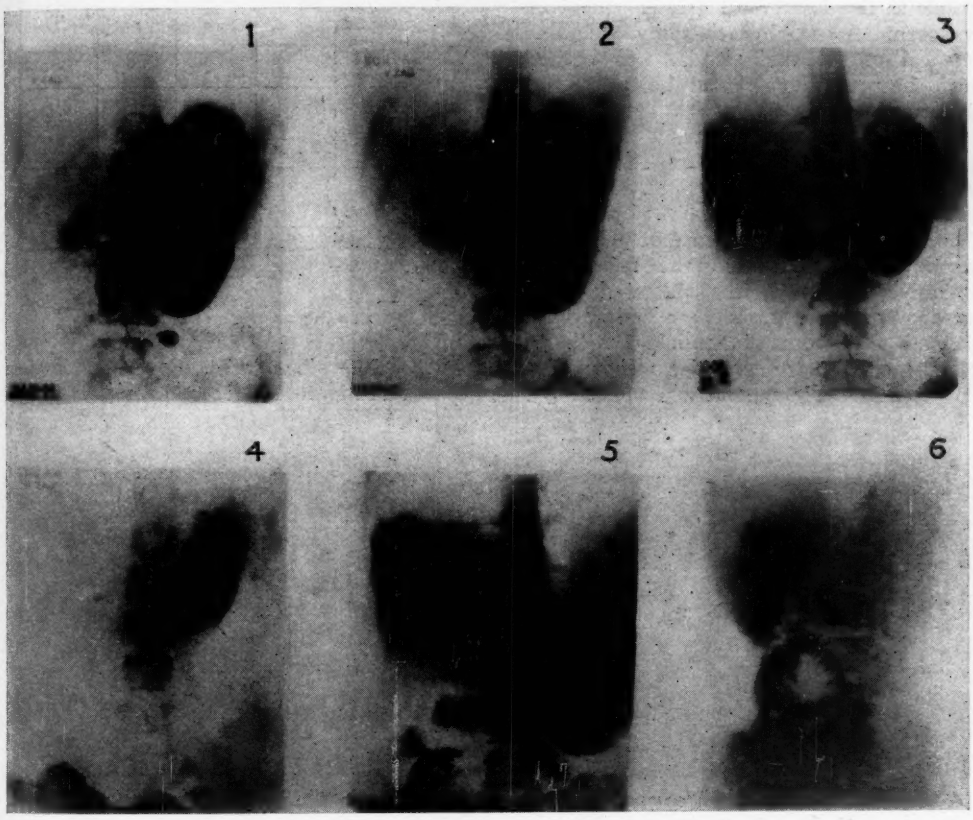

FIG. 266.—*Case* 2. 1—4, Radiographs of barium-meal examination of stomach, showing filling defect. 5, 6, Antero-posterior and lateral views after a posterior no-loop gastrojejunostomy.

PATHOLOGICAL REPORT.—The gland showed chronic inflammatory lymphoid and reticulo-endothelial hyperplasia, tuberculous.

This patient could not be traced, even after repeated attempts.

Case 3.—

HISTORY.—A Hindu male, aged 50 years, a crane driver by trade, was admitted in 1935 for the treatment of pain in the abdomen of fourteen months' duration. For the first eight months the pain was dull and diffuse over the epigastrium and was present all the twenty-four hours of the day without any exacerbations. Subsequently the pain became more severe, and improved after regulation of diet. Later vomiting started. He was constipated and did not give a history of hæmatemesis, melæna, or visible peristalsis.

ON EXAMINATION.—He was found to be emaciated, with a tender spot in the middle line, 2 in. below the ensiform cartilage. The respiratory system was found to be normal. Circulatory system: the first sound was heard feebly in the mitral area. Blood-pressure was

138/90 mm. Test-meal examination showed achylia (*Fig.* 267). Barium-meal examination showed a filling defect in the pyloric antrum (*Fig.* 268, 1–3.). In all the pictures the stomach resembled *Cases* 1 and 2. The Wassermann reaction was negative.

OPERATION.—Under local anæsthesia supplemented with general anæsthesia (ethyl chloride–ether–oxygen sequence), the abdomen was opened, and on investigation a thickened pyloric antrum with enlarged glands at the lesser and greater curvature was seen. A partial gastrectomy, including removal of all the glands, was carried out, the method employed being that of Balfour.

POST-OPERATIVE FOLLOW-UP. — Ten months after the operation the patient reported himself at hospital. He had gained 16 lb. in weight since the operation; there had been no recurrence of pain; the gastric analysis revealed the same pre-operative picture; the blood-chlorides were 548 mg. per 100 c.c.; a barium meal showed the stoma functioning well. A blood-examination revealed no signs of primary anæmia—colour index 0·9; hæmoglobin 80 per cent. The patient was advised to take hepatex and hydrochloric acid for a period of

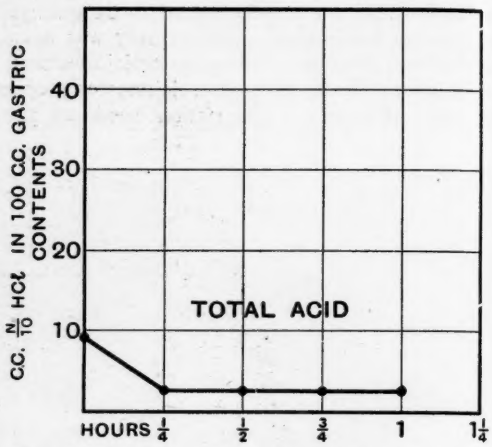

FIG. 267.—*Case* 3. Chart of test-meal examination, showing condition of achylia.

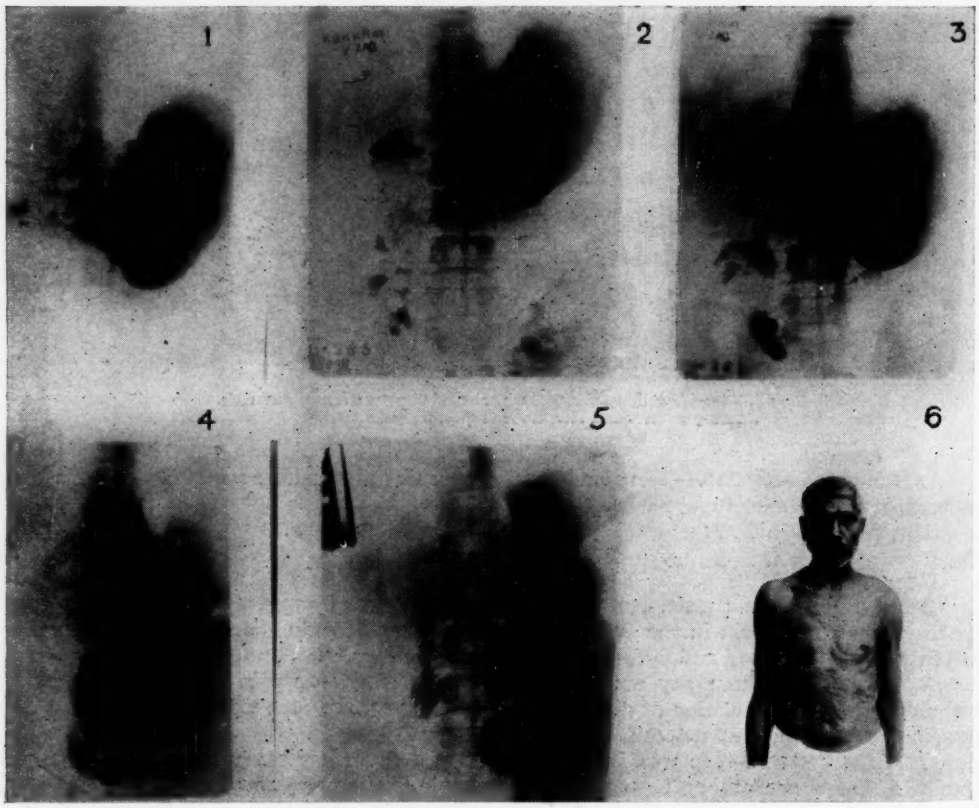

FIG. 268.—*Case* 3. 1—3, Barium-meal radiographs of the stomach, showing the filling defect. 4, 5, Radiographs taken after partial gastrectomy and Balfour's gastro-enterostomy. 6, Photograph of patient six months after operation.

FIG. 269.—*Case* 3. Drawing of the specimen.

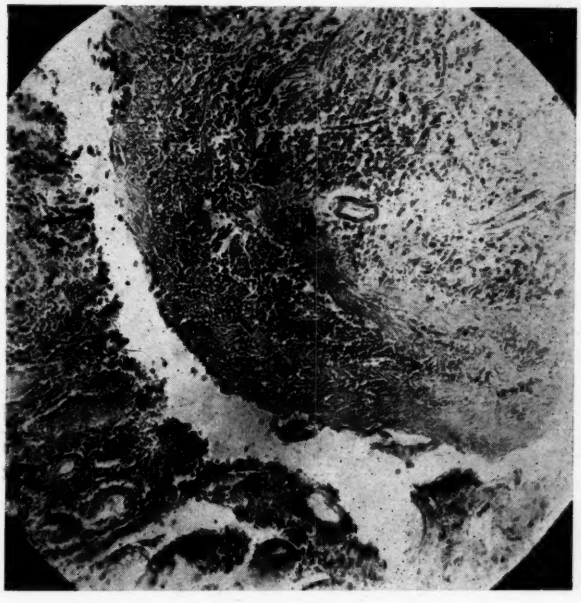

FIG. 270.—*Case* 3. Microphotograph of a section of the specimen.

three months, and asked to report again, or if inconvenient to do so to write a letter regarding his condition. (*Figs.* 268, 269.)

PATHOLOGICAL REPORT (*Fig.*270).—A chronic shallow ulcer of the stomach, with islands of mucous membrane here and there. No evidence of malignancy. The gland shows chronic inflammatory changes; no evidence of malignant infiltration.

Comment.—In this case the pathologist's report did not show even a slight evidence of a tuberculous condition on histological examination. But this was classed as a case of tuberculous ulcer for the following reasons: (1) Gastric analysis showed complete achylia; (2) Macroscopical appearance of the ulcer was the same as in *Case* 1.

My thanks are due to Dr. P. Kesavaswami for the excellent barium-meal pictures and clinical photographs and to the biochemist for his investigations. My thanks are also due to Dr. Tirumurti for his pathological report and to Dr. P. R. Rao for the microphotographs.

SIMULTANEOUS PERFORATION OF MULTIPLE PEPTIC ULCERS

By W. E. AUSTIN, Ashton-under-Lyne

THE LITERATURE

A WARNING is issued in several standard works on surgery that in cases of perforated peptic ulcer a careful search should be made for a possible second perforation. Rowlands and Turner[1] quote Finney[2] as stating in 1900 that a second perforation occurs in 20 per cent of cases, but add that " the coincidence is far less common in the writers' experience ". They also state that " gastric and duodenal ulcers sometimes perforate simultaneously ". Hamilton Bailey[3] makes mention of the fact that they do occur, and that he has seen one case post mortem. Their comparative rarity may perhaps also be judged by the fact that in a general hospital of 200 beds, in an industrial area where perforations are common, two surgeons of wide experience have not seen a case in 25 years.

A careful search of the literature has shown that the number of recorded cases must be small, and that only a few of the patients survived. Masson and Simon,[4] of the Mayo Clinic, surveyed the literature regarding multiple perforated gastric ulcers in 1926. They found 32 authentic cases and reported 1 of their own. They state that " it is only rarely at operation and usually at necropsy that the diagnosis of multiple lesions has been made ". Of the 33 cases 18 had laparotomy performed, but in only 4 cases were both perforations closed; these four survived. There were cases where three or four perforations were present, but these arose from a single large ulcer.

In 1929 Drury[5] published a case of duodenal ulcer with obstruction, which at autopsy was found to have two perforated ulcers of the posterior wall of the stomach. Skemp[6] also recorded a case in 1929.

I have not, however, found an exactly similar case to the one reported below. It is well recognized that gastric and duodenal ulcers are not infrequently associated one with the other in the same patient, but it would seem to be a rare coincidence when they perforate simultaneously.

CASE REPORT

HISTORY.—A man, aged 27, was admitted to the Infirmary, Ashton-under-Lyne, at 5.30 p.m. on January 13, 1937, with acute abdominal pain. The diagnosis with which he came was " acute appendicitis ". He gave a history of a sudden onset of pain at 1 p.m. of the same day. It was severe and mainly epigastric. He had not vomited. He had no frequency or dysuria, and his bowels were regular. He did not volunteer information about having had indigestion, but on careful questioning he said he had had some discomfort after meals since Christmas, i.e., for three weeks, but that he had considered it due to over-eating at the festive season. His previous history held nothing relevant except that he had had all his teeth removed four years before and now had dentures. His habits and mode of life are perhaps of interest. He smoked about 15 cigarettes daily and drank about 2 gills of beer per week. He worked as a cooper at a brewery, but also played in a dance band three nights a week for four hours at a time.

ON EXAMINATION.—He was obviously distressed and held himself rigid. His temperature was 99° F. and his pulse-rate 108 per minute. His tongue was dry and furred. Nothing abnormal was found in his chest. There was board-like rigidity of his upper abdomen, with tenderness most marked in the epigastrium and in the right iliac fossa. Urinalysis showed a normal urine. A diagnosis of a perforated peptic ulcer was made.

AT OPERATION.—Under general anæsthesia a right paramedian incision was made. There was free fluid in the peritoneal cavity. The appendix, being accessible, was examined, found to be rather inflamed, and removed. Attention was then paid to the stomach, and a perforation about 4 mm. in diameter was found on the lesser curvature on the anterior surface, about two inches from the pylorus. This was closed by interrupted Lembert sutures. Obvious free fluid was being aspirated from the peritoneal cavity when it was noticed that some bile-stained fluid seemed to be coming from the right sub-hepatic region. The gall-bladder was examined and found intact, but closer inspection of the duodenum revealed a second perforation on its

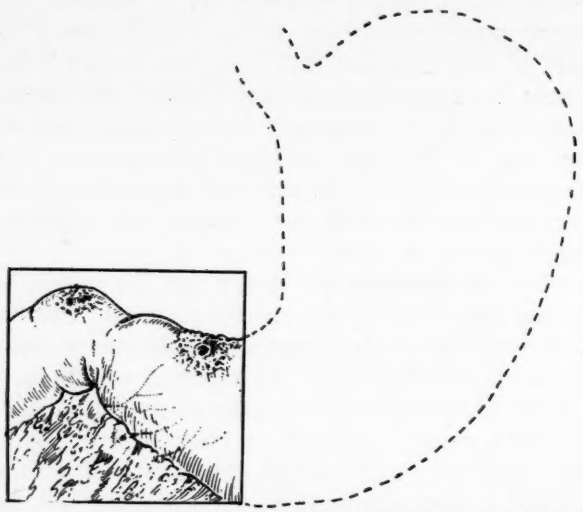

FIG. 271.—Diagrammatic representation of gastro-duodenal region, showing the position of the ulcers, one on the lesser curvature of the stomach and the other on the anterior wall of the duodenum near the pylorus.

anterior surface. This was closed by Lembert sutures and a piece of omentum was tacked down over the area. A suprapubic rubber drain was inserted into the pelvis and the original wound closed in layers. Recovery was uneventful and the patient was discharged home on February 1, on the twentieth post-operative day. (*Fig.* 271.)

SUBSEQUENT PROGRESS.—He was seen again on March 24. He has had no further indigestion or discomfort of any sort. He has adhered to his diet and has gained a stone in weight (he was 11 st. just before operation, and is now 12 st.). As a matter of interest a complete blood-count was carried out, and it was found to be entirely normal. The blood-Wassermann reaction was negative. Radiography with a barium meal revealed only a deformity of the duodenal cap, with no obstruction of the pylorus.

Comment.—It is perhaps worthy of note that the pathological report on the appendix stated that there was "inflammatory changes in the mucosa and submucosa", suggesting that there was a mild inflammation of the appendix apart from a purely peritoneal involvement as a result of the perforations, which were, it will be remembered, of less than six hours' duration.

SUMMARY

A case of simultaneous perforation of gastric and a duodenal ulcer is reported, with recovery after operation. A search through the available literature has shown

that multiple perforated peptic ulcers are uncommon, but do occur. No record was found of a case exactly comparable to the one described.

My thanks are due to Mr. F. G. Ralphs for permission to publish the case and for much encouragement and counsel; to my colleagues, Drs. Walker and Burness, for their assistance with the case; and to Drs. Hartley and Laing for X-ray and pathological reports.

REFERENCES

[1] ROWLANDS, R. P., and TURNER, PHILIP, *The Operations of Surgery*, 8th ed., vol. ii. London: J. & A. Churchill.
[2] FINNEY, J. T., *Ann. of Surg.*, 1900, xxxii, 1.
[3] BAILEY, H., *Emergency Surgery*, 3rd ed., 112. Bristol: John Wright & Son.
[4] MASSON, J. C., and SIMON, H. E., *Minnesota Med.*, 1927, May, 289.
[5] DRURY, R. B., *Ann. of Surg.*, 1929, lxxxix, April, 634.
[6] SKEMP, G. E., and SKEMP, A. A., *Wisconsin Med. Jour.*, 1929, xxviii, Oct., 469.

In Memoriam

SIR DAVID WILKIE, O.B.E.

It may be that it is those of us who knew Wilkie best who find it most difficult to write an appreciation of him. There is so much that might be said, and, being said, it would seem but a fraction of one's thoughts and sentiments. But this feeling is common to all to whom his name was known: that by his passing surgery has lost one of its most eminent figures, one of its most loyal and devoted disciples.

From his birthplace of Kirriemuir, the Thrums of J. M. Barrie, Wilkie came to Edinburgh, and his school years were passed in the Edinburgh Academy.

He graduated at Edinburgh University in 1904, and from that time his rise to distinction was rapid and assured. He held various house appointments in the Edinburgh Royal Infirmary, the Children's Hospital, and Chalmers' Hospital, and it was significant in the light of later events that in each of these he came into contact with men of eminence and distinction in the profession—Dr. Alexander Bruce, Sir Harold Stiles, and Professor Francis Caird. There can be no doubt that their influence helped to mould his ideals and ambitions, and from association with them he gained something of the spirit of their genius in clinical thought and action.

It was the fashion at that time for the young graduate who aspired to specialism to spend a period in study abroad, and particularly in the schools of Germany and Austria. Wilkie followed the accepted course; he spent some time in post-graduate study in Bonn, Vienna, and Berne, and there is no doubt that he retained an admiration for the German surgeon and his work.

On his return to Edinburgh Wilkie became assistant in private practice to the late Professor Caird. It was a happy collaboration, for the wide clinical experience of the older man awakened, and it may be initiated, in the mind of the young colleague an interest in the problems of abdominal surgery. Questions would arise, no doubt, of the why and the wherefore of the progress and treatment of disease. How better could they be answered than by the test of the biological method? And so it was that Wilkie entered upon that line of abdominal research with which his name is so intimately associated. The scope of his interests in these matters was very wide, and there are few surgical diseases of the abdomen which have not come under his criticism and investigation. The value of that work is acknowledged, and it has its reflection in both the pathological and the clinical fields.

His first honorary hospital appointment was to the staff of Leith Hospital, and there he had his introduction to the responsibilities, but at the same time to the pleasures, of an independent charge. From Leith he passed to the wider sphere of the Edinburgh Royal Infirmary, and it was in that institution that he developed those gifts of teaching and of operating for which he was so rightly appreciated and admired. He possessed a wonderful faculty for clinical demonstration; the dullest subject acquired a living interest by his presentation of it, and by the clear and simple logic of his argument he conveyed impressions which could scarcely be forgotten. His skill as an operator was acknowledged by all who

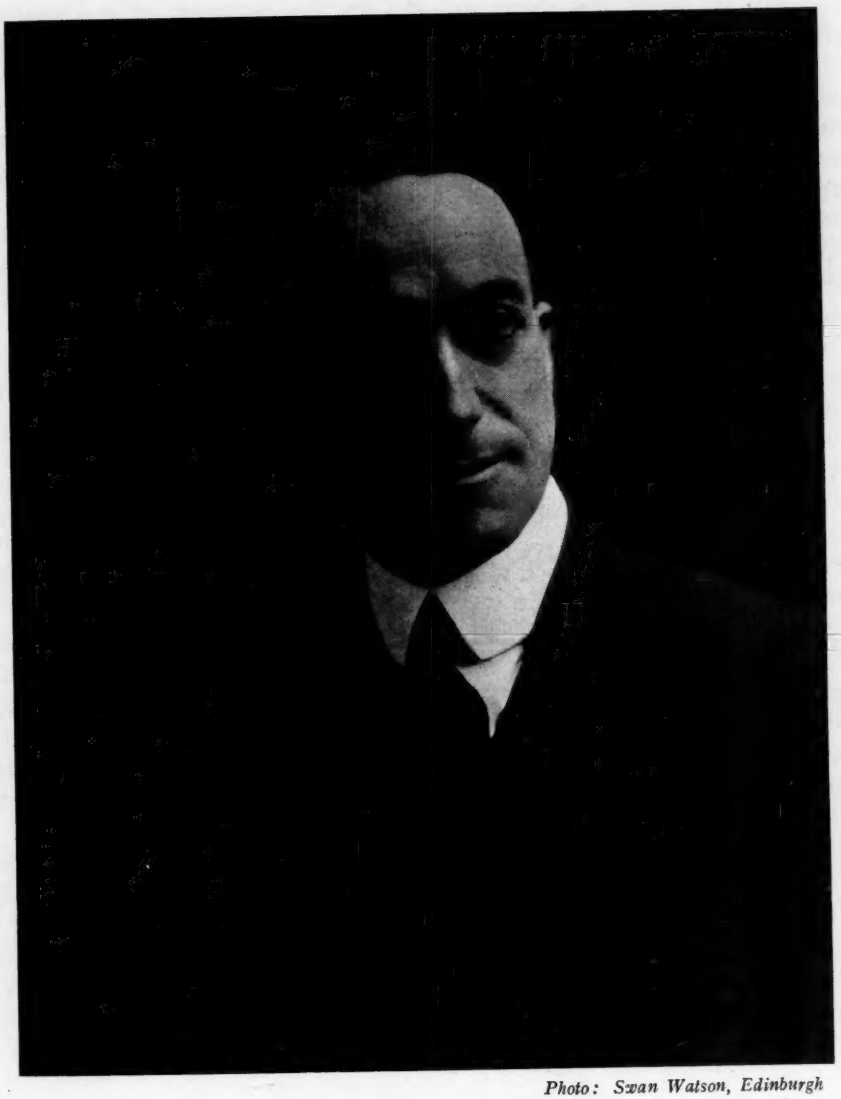

Photo: Swan Watson, Edinburgh

SIR DAVID WILKIE, O.B.E.

1882—1938

witnessed his work. Simplicity was the keynote of his technique, and this, combined with delicacy of touch and sound and balanced judgement, ensured the best possible results.

His appointment to the Chair of Systematic Surgery in 1924 was an appropriate recognition of his merits. The conditions on which the Chair was held had been altered, and its scope had been widened by the inclusion of a Department of Experimental Surgery. This gave him an opportunity for developing his ability as an administrator, while at the same time it afforded him a unique opportunity for guiding and controlling the research activities of his younger colleagues. The scheme was a success from the first; Wilkie gathered round him a band of loyal and devoted workers, and in the fourteen years which have elapsed since its inauguration the department has produced much interesting and significant research.

He added yet another to his heavy responsibilities when he accepted the post of Director of the Surgical Unit of the municipal hospitals. He assumed this further burden because he foresaw the possibilities of future development; a link between the municipality and the University had been created in relation to hospital work and clinical teaching, and Wilkie recognized that this association, if rightly guided and controlled, might react beneficially on both partners of the agreement. He wrought unceasingly in this field, and no doubt the fruits of his labours are apparent.

With so many interests of an administrative character in his hands, it was natural that he should act on the body through which University decisions are made, and in 1935 he was appointed to the University Court as a representative of the Faculty of Medicine.

For any ordinary man such multifarious activities would surely have filled the cup full to overflowing, but here was no ordinary individual: it seemed as though Wilkie's energy was boundless and his interests without limit. A multitude of good causes claimed his attention and support: he was Chairman of the University Settlement, and the donor of the Kirk o' Field College, an educational institute for those to whom opportunity is otherwise denied; he was a Director of the Edinburgh Medical Mission, and Chairman of the Edinburgh Committee of the Moukden College; and these were only part of his activities. His scientific interests were linked with the Medical Research Council, the Scientific Committee of the British Medical Association, and the British Empire Cancer Campaign, besides others too numerous to mention. The field amazes one; that any single individual could undertake it all! And yet he did so. It was only possible to one with superb organizing ability, and this Wilkie possessed in a marked degree. And with it all he went calm and unruffled through life; the honours and distinctions which were bestowed upon him he carried with a modesty which all admired, and if he encountered troubles and worries, as no doubt he did, he covered them with a cloak of peace and courage that testified to the strength and stability of his mind.

There is much more that might be said—of his war service in the navy, of nis interest in the student body, of the attractiveness of his personality, of his kind and courteous attitude to all with whom he came in contact. These things are known to those with whom he was associated, and they are among the memories which they will recall when his name is mentioned. He was a worthy son of Scotland, and when he was knighted in 1936 the land of his birth was proud that this well-merited distinction had been bestowed upon him. His memory will remain bright and untarnished, and his spirit will beckon towards those things that are worthy and true.

CRITICAL REVIEW

THE SURGICAL TREATMENT OF OBSTRUCTIVE JAUNDICE IN PANCREATIC DISEASE*

By Sir JOHN FRASER, K.C.V.O.

INTRODUCTION

In November of last year I was asked by the Council of the Association of Surgeons if I would open a discussion on "The End-results of the Surgical Treatment of Obstructive Jaundice in relation to Pancreatic Disease". I accepted the invitation with much pleasure, but I felt that, if the discussion was to be of value, it would be necessary to prepare a sufficient body of evidence to present a reliable appreciation of the problem. It has been said on many occasions that statistics are but a pitfall and a snare, and that by judicious use of them utterly divergent views may be buttressed and supported; that, in fact, anything can be proved by them. I daresay there is some truth in the statement, but it seems to me that the criticism weakens as the volume of statistics increases. The risk lies in drawing conclusions from relatively small numbers, and therefore it was to obviate that risk that during these past months I have troubled you with requests for facts and figures and information. Two hundred and two inquiries were sent out, and it was encouraging to receive one hundred and seventy-two replies. I take this opportunity of thanking those who have been so kind as to respond to my inquiries, and I wish to assure them that if the report has any value it is attributable to the co-operation of the Fellows of the Association of Surgeons.

I assumed that when the Council chose this subject for inquiry they anticipated that it might yield interesting and suggestive results. I do not think that it was expected that the results would be encouraging, but it would be unwise to quarrel with their philosophy on that account, for surely it is better to know the truth, even if it be unwelcome, than to live in a fool's paradise. It may be that, knowing the worst, we may be able to obtain some guidance as to how matters can be improved. I have done what I can to sift the information I have received, and to present the facts which emerge from it.

Obstructive jaundice, the result of pancreatic disease, is not a common condition. Coughlin and McCaughan[1] reported 74 cases in 76,000 admissions to hospital, and these figures appear to indicate a fairly accurate proportional representation of the position. Let us put it at 1 in 1000 admissions, and we are probably not far from the truth. It is apparent that it is difficult for any single individual to come into contact with a sufficient number of cases to make the results conclusive or reliable, but by the system of 'pooling' our experiences a total of 1035 cases has been placed at our disposal.

* A collective inquiry into 1035 cases instituted by the Association of Surgeons and reported on at the London Meeting, May 6, 1938.

I realize that the form of questionnaire which was sent out was incomplete, but it was necessary to keep its length within bounds, and I think it has succeeded in eliciting the bulk of the essential facts. Many contributors took advantage of the last section, that which asked for suggestions as to how future results might be improved, and under this heading a number of interesting details and comments were offered.

I shall summarize the position as far as such is possible, but the manner in which I have chosen to debate the problem demands a short explanation. As the title stands it might be possible to dismiss the subject quite briefly, and, with the aid of a few statistics, to present figures which would illustrate reasonably accurately the end-results of the surgical treatment of obstructive jaundice in pancreatic disease. I fear, however, that this would be but a dry business, and a depressing one at that, and therefore I have thought it better to widen the scope of the discussion to a greater degree than the title would imply, and to touch upon questions outwith the realm of pure statistics—matters that concern the diagnosis, the pathology, and the treatment.

If a review of this kind is to be worth while, it should offer two results : a fair assessment of the existing position ; and the provision of information which may hold out a hope of future improvement. I feel that to widen the discussion is the means by which we are likely to attain these ends.

THE TOTAL CASES : TYPES AND PERIOD

As I have said, details of 1035 cases were given, and all are true to type in the respect that they were examples of obstructive jaundice arising in relation to pancreatic disease. There are, however, two qualifications to which I must allude. I have restricted my review to examples of chronic disease, and therefore I have omitted from the records acute pancreatitis and a few cases of chronic pancreatitis associated with gall-stones, in which the element of obstructive jaundice was doubtful. Therefore all the cases under consideration represent obstructive jaundice secondary to chronic pancreatic disease. The second qualification is in respect of the dates over which the cases have been observed. The overwhelming majority were treated in the decade from 1927 to 1937, and we may assume that they benefited from modern ideas regarding the diagnosis and treatment of the disease.

THE SEX AND AGE ANALYSIS

In respect of sex, the incidence of males exceeds females in a proportion of 2 to 1, the actual figures being 682 males and 353 females.

The age occurrence covered a wide period, ranging from 34 to 82 years, with a peak incidence in the decade from 55 to 65 years. It was interesting to note the constancy of this occurrence, and I suppose it is in keeping with the accepted facts of chronic disease incidence. The analysis is in respect of the total cases.

As a matter of interest I attempted a contrast analysis between the age-incidence of malignant cases and chronic pancreatitis (*Fig.* 272). As the graph shows, there is comparatively little distinction ; chronic pancreatitis has a more restricted age ambit, but its maximum occurrence is very similar to that of malignant disease.

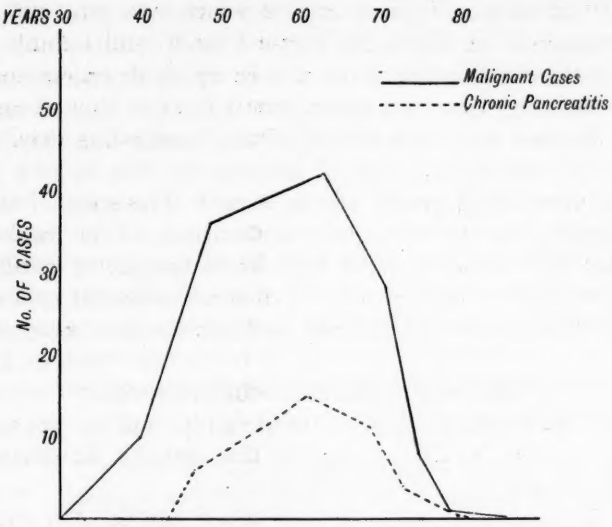

FIG. 272.—A graph representing the respective age-incidence periods of malignant disease of the pancreas and chronic pancreatitis.

THE TYPES OF PANCREATIC DISEASE

The Difficulty of Distinguishing Between Simple and Malignant Conditions.—*Table I* records the various types of pancreatic disease responsible presumably for the obstructive jaundice.

Table I.—TYPES OF PANCREATIC DISEASE

DISEASE	No. OF CASES	PERCENTAGE
Carcinoma of pancreas (disease restricted to head of gland)	694	67·0
Carcinoma of pancreas (diffuse disease)	62	5·9
Local tumour described as enucleable	4	0·38
Tumour described as pancreatic cyst	12	1·16
Chronic pancreatitis	238	23·0
Pathology not stated	25	2·4
TOTAL	1035	

I encountered some difficulty in arriving at this analysis, and the figures, though reasonably accurate, must be regarded as provisional.

It was exceptional to find the diagnosis based upon biopsy. There are some cogent objections to incising a pancreatic tumour, and it is apparent that this is the majority opinion in an overwhelming degree. It follows, therefore, that in most instances the diagnosis was based upon the morbid anatomy and upon the degree of hardness of the gland when it was submitted to palpation examination. This feature is reflected in the following statement which was typical of that made by a number of observers: "I found it impossible to decide at operation whether the tumour was inflammatory or malignant, but, in view of the fact that the patient has survived over an appreciable period of time, I have no doubt now that the disease was in the former category."

It is evident that such an opinion expresses a real practical difficulty, and there must be many occasions on which the surgeon finds it impossible at operation to state dogmatically the exact nature of the disease which he encounters. This review has revealed the difficulty time and again, but it has not elicited any facts of value in coming to a decision as to the nature of the border-line case, although it has brought to light the interesting piece of information that in 16·8 per cent of the cases the morbid anatomy diagnosis was in error. The state of the pancreas was regarded as malignant, but the subsequent survival-rate of the cases on a two-years' basis showed that the condition must have been simple. I allude to this point because it seems to indicate that, if there is doubt, the benefit of it should be given to the simple possibility, and steps should be taken to afford operative relief.

PRE-OPERATIVE DIAGNOSIS

THE EVIDENCE UPON WHICH IT WAS BASED

If there was difficulty in recognizing the morbid anatomy at the time of operation it seems logical to inquire as to the pre-operative position. Was the existence of a pancreatic lesion as the source of the obstructive jaundice arrived at before operation, and, if so, what were the facts upon which the decision was made? I know that this is quite beyond the remit of my instructions, but the opportunities afforded are so unique that I think we should seize the opportunity to learn what we can from the great mass of material available.

I have studied this aspect of the question as closely as possible, and here is a summary of my impressions. Let me take matters in a logical sequence.

The Presence of Jaundice.—Jaundice was present in every instance; it was the uniform feature which justified the inclusion of the case, and as a matter of general interest its degree was inquired into. In the questionnaire the terms 'slight', 'moderate', and 'severe' were used; for these I have substituted Grades I, II, and III, and I have prepared a table which reviews the grading results in 974 cases representing all types of disease.

Table II.—The Degree of Jaundice in All Types of Pancreatic Disease

Grades	*No. of Cases*
I	38
II	300
III	636
Total	974

This table as an independent statement has no particular value, but I have carried out a further analysis which appears to yield more informative results. I have analysed the jaundice gradings in malignant and in non-malignant cases respectively, and I now present the results (*Tables III, IV*).

Table III.—Jaundice Grading in Carcinoma of the Pancreas

Grades	*No. of Cases*
I	—
II	139
III	568
Total	707

Table IV.—JAUNDICE GRADING IN CHRONIC PANCREATITIS AND OTHER NON-MALIGNANT CONDITIONS

Grades	*No. of Cases*
I	38
II	162
III	67
TOTAL	267

I believe that these tables have some interest; they afford definite evidence that the malignant type of pancreatic disease is the one which is associated with the more severe degrees of jaundice—in other words, it may afford the surgeon the first clue regarding the nature of the pancreatic disease.

The Obstructive Nature of the Jaundice.—Accepting the fundamental fact of the jaundice, the next step was to recognize its obstructive character. The history, the associated physical condition, and above all the severity of the sign were generally sufficient to establish obstruction characteristics, but in many instances the van den Bergh biochemical test was adopted, and it is a testimony to the accuracy of this investigation that, employed in 578 instances, it yielded an immediate direct reaction in every case. The inquiry elicited much interesting information regarding the results of the reaction, particularly in connection with the value of biphasic readings as an indication of liver damage, and therefore of value in prognosis. I allude to this point later.

The Pancreatic Source of the Obstruction.—These, then, were the two preliminary observations—jaundice was definite, and it was frankly obstructive in character; but was the obstruction pancreatic in source, and, if so, was there evidence to indicate the nature of the pancreatic obstruction?

As far as I have been able to judge, the facts which led to a pre-operative decision on these points were as follows:—

1. *The Sequence of the History (Distinction between Simple and Malignant Cases).*—A number of the reporters were so good as to place complete case histories at my disposal, and I had an opportunity of correlating and comparing these. I imagine it is agreed that the history is probably the most important item in suggesting the pancreatic locus of the obstruction; but I would go further, for a study of these records appears to indicate that a distinction in sequence is apparent between the malignant and the simple case. My impression is that in the malignant cases there is nausea and anorexia, an early loss of weight, and the appearance of cachexia, and that these signs always precede the development of the jaundice. On the other hand, if the lesion is a simple one—a chronic pancreatitis, a simple tumour, or a cyst—while there may be indefinite complaints of upper abdominal discomfort, jaundice is usually the first indication that something significant is at fault. I do not say that the distinction is a constant feature, but it was recorded with sufficient frequency to imply that mention should be made of it.

2. *Pain—Its Incidence and Characteristics and the Reaction to Morphia.*—It is sometimes said that a painless jaundice is characteristic of a pancreatic lesion. The records of this inquiry do not support this statement. Pain was recorded in 54 per cent of the cases, it was described as 'dull', 'constant', and 'aching', and it was indicated as occurring to the right of the epigastrium and beneath the angle of the scapula. Of more significance, however, was the fact that colic of a biliary type

was recorded in 12 per cent of the cases, though subsequent operation made it clear that no gall-stones existed. This is an important observation, but at the same time an unfortunate one, because it is apparent that this occurrence must tend to obscure the diagnosis.

Arising out of it there is a point to which I feel I ought to allude, as it may have practical importance. Several observers noted that when the colic developed it was not relieved by morphia, but in fact tended to be increased by this drug. I have been reminded that the experimental work of McGowan, Butsch, and Walters[2] may support this observation and afford its explanation. You may recall that they investigated the pressure in the common bile-ducts of patients whose gall-bladders had been removed, and they found that the administration of morphia produced a rise of pressure in the common bile-duct to such an extent that pain was produced. It may be that these experimental observations have a parallel in the distended gall-bladder and bile-ducts associated with pancreatic disease, in which event it is possible that the response to the administration of morphia may form an aid in the diagnosis.

3. *The Physical Examination.*—

The state of the gall-bladder: Apart from the jaundice and the characteristic general appearance of the patient, the condition of the gall-bladder calls for comment, and, as changes in the organ may be perceived on abdominal examination, the findings in relation to it are worth noting. A distended and tense gall-bladder was noted in 88 per cent of the malignant cases. Where enlargement was not apparent, it was noted that the organ was the site of a chronic cholecystitis, but even so it was tense, though its walls might be thickened and contracted. It is evident, then, that distension of the gall-bladder was an almost constant finding in malignant pancreatic disease.

The position was different in chronic pancreatitis; in 15 per cent of cases it was described as being tense and distended, but in the remaining 85 per cent of cases inflammatory disease of the organ with or without gall-stones was recorded, and, though the organ was tense, it rarely attained the size encountered in association with malignant disease of the pancreas.

These findings, apart from their diagnostic value, seem to indicate the significant part which preceding gall-bladder infections play in the production of chronic pancreatitis.

In the sixteen examples of simple pancreatic disease the comment was made that the gall-bladder and ducts showed moderate distension without evidence of previous disease.

THE VALUE OF BARIUM MEAL EXAMINATION

In a small number of cases (37 to be exact) an X-ray barium meal examination was carried out, but the results were not sufficiently characteristic or constant to be of real value. In a few instances suggestive findings were recorded in the second part of the duodenum—fixation, distortion, and dilatation, but it is evident that this method of examination has no value as a diagnostic agent.

To sum up the position at this stage, the records appear to indicate that a provisional diagnosis of pancreatic disease being responsible for the jaundice was arrived at by: (1) The history; (2) The general physical condition of the patient; (3) The state of the gall-bladder as revealed on abdominal palpation.

It is noteworthy that the pancreatic tumour was rarely detected by palpation; it is said to have been detected in 17 instances, and 10 of these were afterwards classified as pancreatic cysts.

THE VALUE OF SPECIAL TESTS

We have arrived at a stage where, a provisional diagnosis having been made, *special tests* were carried out, and I propose to give a brief consideration to their results and values.

I asked myself why they were carried out, and the answer appeared to be: (1) To confirm the obstructive character of the jaundice; (2) To estimate the degree of the obstruction; (3) To suggest the site and the extent of the obstruction; (4) To indicate the effect of the obstruction on vital functions.

Perhaps you will allow me to summarize certain points in relation to each:—

1. Confirmation of the Obstructive Character of the Jaundice.—I have already alluded to this point, and I have described the efficiency of the van den Bergh reaction as practised in 578 cases. Some observers have expressed the opinion that a biphasic reaction is a reasonably accurate indication of liver damage, and that it is therefore of value in estimating the prognosis. On the other hand, there is an opposite opinion, and definitely the majority one, that a biphasic reaction has no particular significance. The evidence obtained by the inquiry is not sufficiently complete to justify a definite conclusion.

The presence of bile in the urine and its absence from the stool has been a general observation in obstructive jaundice, but a number of reporters have drawn attention to the fact that in simple chronic pancreatitis, while the jaundice may be profound and while abundance of pigment is evident in the urine, bile may exist in the stool to an appreciable degree. There are some who go so far as to say that the evidence is of value in distinguishing between the simple and the malignant pancreatic lesion.

2. An Estimate of the Degree of the Obstruction.—

The Value of the Icteric Index and Serum-bilirubin Readings.—The importance of estimating the degree of jaundice has been alluded to in a clinical sense when reporting the findings in relation to the intensity of the jaundice, and the majority of those who have replied to the questionnaire have expressed the view that the simple clinical observation is sufficient indication of the degree of the obstruction, but in a number of instances biochemical tests have been employed with a view to expressing the degree of obstruction in a more exact manner and by means of figures. The tests employed have been the *icteric index* and the *serum-bilirubin* estimate. When these tests were applied with any degree of regularity, appreciation of their value was expressed, but it was pointed out that to understand their full significance they should be repeated daily or even twice a day. Under such conditions they afford not only an accurate estimate of the degree of obstruction, but, if they show a rapid and progressive rise in their intensity, the finding implies an approaching liver failure, and therefore increasing anxiety in respect of prognosis.

The icteric index figures ranged from 18 to 262, and it was agreed that any reading above 150 was ominous. The serum-bilirubin readings suggested parallel conclusions. Under normal conditions the estimates range from 0·1 to 0·5 mgm. per cent; the readings were recorded in 123 cases, they ranged from 2·5 to 14·6 mgm. per cent, and it was agreed that any figure in excess of 5 mgm. per cent

appeared to be associated with definitely increased risks. I have attempted to tabulate these impressions in so far as they apply to the icteric index in a simple graph (*Fig.* 273). I do not claim that it is an accurate representation of the position, because I found it difficult to extract exact comparisons between icteric index and

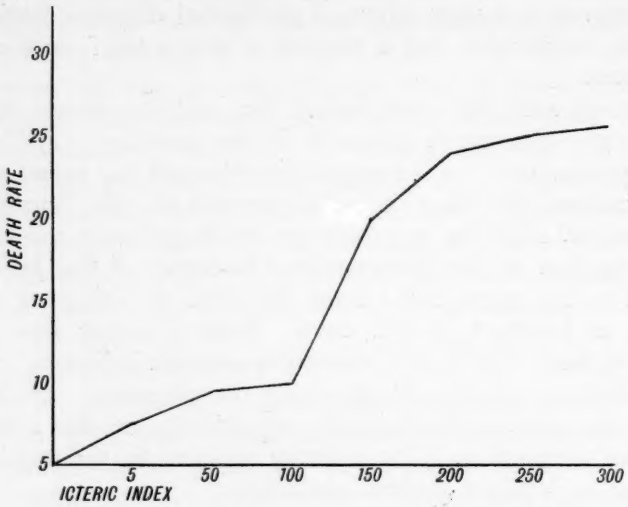

FIG. 273.—Graph to illustrate the association between icteric-index readings and mortality-rate. Note the rapid rise in mortality-rate when the icteric index exceeds 100.

serum-bilirubin readings on the one hand and mortality figures on the other, but the chart has been based on what records I could find, and at least it represents in a graphic way the conclusions of a number of the reporters.

3. The Site and Extent of the Obstruction.—

Tests Revealing the Degree of Pancreatic Involvement.—In a small series of these cases (42 in number) an investigation was made which opens up interesting possibilities—namely, investigation of the stool for evidence of interference with the pancreatic external secretions.

It will be recalled that in pancreatic insufficiency the total fat of the stool is increased up to 70 or 80 per cent, and the increase is found to be in the neutral fats, being the result of defective fat splitting. In pure obstructive jaundice, which does not imply interference with the external secretion of the pancreas, the total of fat is also increased, but, since the fat-splitting action is normal, the increase is in the fatty-acid fraction. This line of investigation has been pursued in 42 cases, and the results suggest that it may afford a means of recognizing the class of case in which the obstructive jaundice is associated with pancreatic malignancy, for in the latter both the neutral fat content and fatty acids of the stool are increased, while in chronic pancreatitis the increase is mainly in the fatty acids. I mention this point because it was the only evidence brought out in the investigation which seemed to offer the possibility of an accurate pre-operative diagnosis in respect of the type of the pancreatic affection.

4. The Effect of the Obstruction on the Vital Functions.—

Sugar-tolerance and Blood-urea Tests.—It will be agreed that, if evidence could be obtained that the biliary obstruction was resulting in serious crippling effects

upon vital structures, the information would be of great value in prognosis, in deciding upon the suitability of individual cases for operation, and in indicating the necessity for pre-operative therapy. The records revealed that two tests were employed with this purpose in view, the lævulose or galactose tolerance test and blood-urea estimation. The first test was employed to determine the efficiency of liver function; the second was intended to explore the possibility of renal damage.

I think it will be agreed that the adoption of these tests is both justified and of value. Much work has been done in these past years on the cause of death in obstructive jaundice, and, while operation has its own peculiar risks, it is evident that the essential lethality of the condition is related to a sequence of events which culminates in one or other of two disasters—liver death or renal death. It seems likely, therefore, that tests designed to establish the physiological condition of liver and kidneys in the presence of obstructive jaundice would have obvious advantages. Both have been employed; not in large measure, I agree, but definite opinions have been expressed upon their value, and I feel it is an obligation to report them.

Those who employ the *sugar-tolerance test* regard it as a reliable indication of the state of liver function, but it has been pointed out that, if the findings are to be relied on, blood-sugar estimates must be done, urine estimations not being sufficiently constant to be of value.

The *blood-urea estimation* was reported in a reasonably large number of cases, and the impression of those who practised it may be summarized as follows. In the later stages of obstructive jaundice the blood-urea content is invariably raised, the increment reaching its highest level a few days after the decompression of the biliary tract has been carried out, and there follows the statement, which of course is complementary to the other, that high blood-urea readings imply an increased gravity in the prognosis.

Blood-coagulation Tests.—The only other pre-operative test which calls for comment is the estimation of the blood-coagulation period. Abundant figures on this point have been supplied, but it is interesting to notice that they do not reveal a striking departure from the normal. Average coagulation times are liable to show considerable variability, and it has been estimated that anything from two to eight minutes may be regarded as physiological. The figures recorded in obstructive jaundice of varying sources ranged from three to nine minutes. It is true that in the most severe examples of jaundice the greatest amount of delay was noted, but one wonders if the danger of delayed coagulation has not been over-estimated.

TREATMENT

For and Against Operation.—There is a strong divergence of opinion on one vital question, Should obstructive jaundice, the result of malignant disease of the pancreas, be operated on? I quote a view which expresses one line of thought: "If I operate on these cases they die in three weeks; if I leave them alone they die in three months, and I believe therefore that it is unjustifiable to submit them to the immediate risks and the subsequent discomforts of a surgical operation."

In contrast to this I recall the opinion of one who, though he has not contributed to this review, claims our attention and our respect—I refer to the late Lord

Moynihan.[3] He wrote: "No one living is infallible in the differential diagnosis of obstructive jaundice. The diagnosis is always so difficult and the chances of a life saved so important that, however positive the evidence of malignancy may be, I now advise operation in all cases . . .

"Apart altogether from the prolongation or saving of life, almost every patient will declare that the relief from the intolerable torture of itching is worth every sacrifice. I suspect that the mortality from suicide in this disease is greater than that from the operations which afford relief."

Thus there are two opinions, utterly divergent, and in their respective points of view equally supported by argument and ethics. If this inquiry has no other value, you will agree, I think, that it will be justified if it enables us to decide which of these attitudes is the more reasonable.

Numerically the opinion revealed in this inquiry is strongly in favour of operation. I received 172 replies to the questionnaire which I sent out; 4 were against operation, 168 practised it. The inquiry should enable us to decide which attitude is correct, and when I have presented the results you may be able to discriminate, but I would say now that the crux of the whole matter is accurate diagnosis. It is so difficult to be dogmatic about the cause of the obstruction; history may be misleading, tests are at the best uncertain, and even the final court of appeal, a laparotomy, may leave the position undecided. In the presence of so much uncertainty it is likely that operation will continue to be practised; and, if so, it seems reasonable that, having ventured so far, an attempt should be made to give relief, even in the malignant case.

Pre-operative Care.—All the reporters were satisfied of the importance of pre-operative care. Intravenous infusions of glucose and calcium were particularly favoured. Pre-operative blood transfusion, a precautionary measure once so popular, was employed in a surprisingly small proportion of cases (4 per cent), and the intramuscular injection of 30 per cent sodium citrate immediately before operation was mentioned on three occasions only. One surgeon employs as a routine measure the withdrawal of 10 c.c. of blood from the patient's arm vein and its re-injection intramuscularly. He apparently is satisfied of the value of this simple procedure.

These various means have two purposes in view: to reinforce a failing liver function and to lessen the risk of post-operative hæmorrhage. Their adoption was so universal that there was comparatively little 'contrast' material, and therefore it is difficult to arrive at an estimate of their efficacy or reliability.

The Types of Operation: Their Values and Risks.—I have tabulated the various operations which were practised in 863 cases. It is an interesting list, and it certainly presents a wonderful variety of procedures!

It is a general list; that is to say, it presents the procedures carried out in different types of pancreatic disease, the common factor being the implication that relief should be afforded from the obstructive jaundice, but there are one or two points about it which call for explanation and for comment.

The operations may be divided into three classes: (1) Internal drainage; (2) External or surface drainage; (3) Two-stage operation.

The operation of pyloric exclusion associated with cholecyst-jejunostomy is in a class by itself; it is intended to be a preliminary to removal of a pancreatic tumour. The indications for it must be vary rare, and it does not enter into our

present considerations. For obvious reasons the exploratory operation is outwith this discussion, and we are therefore left with the three standardized types which I have already mentioned.

Table V.—TYPES OF OPERATION

Operation	*No. of Cases*
Cholecyst-gastrostomy	397
Cholecyst-duodenostomy	144
Cholecyst-enterostomy	67
Cholecyst-colostomy	5
Choledocho-gastrostomy	3
Choledocho-duodenostomy	11
Surface drainage operation (cholecystostomy and choledochotomy)	84
Two-stage operation (cholecystostomy and cholecyst-gastrostomy)	57
Pyloric exclusion and cholecyst-jejunostomy (in anticipation of removal of pancreatic tumour)	2
Exploratory operation	93
TOTAL	863

Internal Drainage Operations.—Of these operations (627 in number) cholecyst-gastrostomy was that which found greatest favour. It was practised in 397 cases (63·3 per cent), and these figures are sufficient indication of its popularity. It is natural to ask why it should be so manifestly the choice, and I imagine that there are two reasons: the contiguity of the viscera; and the belief that a cholecystogastric fistula approximates to physiological conditions.

Those who do not practise it have not hesitated to express criticisms of it. They claim that the anastomosis is liable to leak owing to the tension created by movements of the stomach, and they point out that, if the anastomosis is completed close to the pylorus, there is interference with the emptying of the stomach and a tendency to the subsequent development of pyloric obstruction. The reports which I have read seem to lend support to the latter objection. In 541 operations implying anastomosis of the gall-bladder to the stomach or duodenum the necessity for a subsequent gastro-enterostomy arose in 23 instances (4·2 per cent of the cases). I daresay that in some of these cases the obstructing element was an extension of the tumour into the duodenal wall, but this was the exception, and in the majority of the cases it was noted that a simple stenosis at the point of anastomosis constituted the obstruction.

Those who practised cholecyst-enterostomy (67 cases) were satisfied of the advantage of this operation, and when I present the mortality statistics it will be seen that there is statistical support for their satisfaction.

I was interested to come across two records of cholecyst-colostomy, and we have the assurance of those who practised such operations that they can be carried out without any evident derangement of intestinal function.

Surface Drainage Operations.—These operations (84 in number) were mainly restricted to cases of chronic pancreatitis, and, as far as I have been able to judge, the results justified the choice.

Two-stage Operation.—Perhaps the greatest interest, however, centres round the two-stage operation—cholecystostomy followed at a later date by cholecyst-gastrostomy or enterostomy. It is evident that two considerations have influenced those who practised this method: the desire to re-establish liver function as a preliminary; and subsequently to complete an anastomosis under conditions of

greater security than is possible in a single-stage procedure. It is my impression that there is much to be said for these arguments, and the immediate results, as far as mortality is concerned, seem to justify the plea.

The Mortality-rate of the Various Operations.—The touchstone of success in any operation, however, is the mortality figure with which it is associated. *Table VI* apportions the hospital mortality of the different procedures, and when I use the term ' hospital mortality ' I imply death within a period of six weeks after the operation.

Table VI.—Operation Mortality-rates for all Classes of Pancreatic Disease (768 Cases)

Operation	No. of Cases	Hospital Mortality
		Per cent
Cholecyst-gastrostomy	397	34·8
Cholecyst-duodenostomy	144	37·6
Cholecyst-enterostomy	67	29·6
Cholecyst-colostomy	5	?
Choledocho-gastrostomy	3	75·0
Choledocho-duodenostomy	11	69·0
Surface drainage operation	84	11·6
Two-stage operation	57	24·0

This table permits us to draw one general conclusion: when operation is carried out for obstructive jaundice arising in association with pancreatic disease the average hospital mortality is 40·1 per cent. A further general comment which may be made is that the safest operation is a simple surface drainage (11·6 per cent mortality), but against this one has to put the fact that it is but a temporizing expedient, which, if it persists, is associated with serious disadvantages.

Of the internal drainage operations, anastomosis of the duct incurs the heaviest mortality (72 per cent in 14 cases), cholecyst-duodenostomy shows a slightly higher mortality than cholecyst-gastrostomy, while cholecyst-enterostomy is appreciably safer. Two-stage operations, as I have indicated, appear to present a reasonably good prognosis in a situation which is always serious, for their mortality-rate is 24 per cent.

Operation Mortality Figures in Malignant and Non-malignant Cases.—Let us now consider the question on a more selective basis, dividing the cases into two main groups: (1) Malignant disease of the pancreas; (2) Chronic pancreatitis.

In *carcinoma of the pancreas* the operative procedures are shown in *Table VII.*

Table VII.—Operation Types in Malignant Disease of the Pancreas (633 Cases)

Type of Operation	No. of Cases	Incidence
		Per cent
Cholecyst-gastrostomy	328	51·81
Cholecyst-duodenostomy	97	15·32
Cholecyst-enterostomy	44	7·0
Cholecystostomy	48	7·55
Laparotomy	55	8·69
Miscellaneous	8	1·26
No operation	53	8·37

The results of these operations are shown in *Table VIII.*

Table VIII.—Mortality Incidence in Malignant Disease of the Pancreas (407 Cases)

Result	No. of Cases	Percentage
Died in hospital from operation	211	51·84
Died in hospital from intercurrent disease	7	1·72
Discharged from hospital, later reported dead	119	29·23
Discharged from hospital, still alive	21	5·16
Discharged from hospital, no further record	49	12·04

In *chronic pancreatitis* the operation incidence and distribution were as shown in *Table IX.*

Table IX.—Operations Carried out for Chronic Pancreatitis (226 Cases)

Type of Operation	No. of Cases	Incidence
		Per cent
Cholecyst-gastrostomy	102	45
Cholecyst-duodenostomy	41	18
Gall-bladder and common bile-duct drained	32	14
Cholecystostomy followed by cholecyst-duodenostomy	12	5
Cholecystectomy with drainage of common bile-duct	9	4
Cholecystectomy	19	9
Laparotomy	9	4
No operation	2	1

The results which followed are summarized in *Table X.*

Table X.—Mortality Incidence in Chronic Pancreatitis

Result	No. of Cases	Incidence
		Per cent
Died in hospital from operation	45	19·8
Died in hospital from intercurrent disease	3	1·3
Discharged from hospital, later reported died	37	16·3
Discharged from hospital, still alive	135	59·5
Discharged from hospital, no further record	7	3·0

Such is the position in respect of operation, and the vital point which emerges is that in malignant disease of the pancreas there is a direct operative mortality of 51·8 per cent, while in chronic pancreatitis the corresponding mortality figure is 19·8 per cent. This latter figure is higher than I had anticipated, and I imagine that there are many who will be astonished to learn that the operative mortality-rate in chronic pancreatitis associated with obstructive jaundice is virtually 1 in 5. It is certainly vastly better than the mortality-rate in malignant cases, which is 1 in 2, but when one realizes how different are the local conditions, it seems to me that we might have anticipated an even more encouraging figure in relation to the

simple condition. The figures are an evidence of the serious position created by an obstructive jaundice.

Factors which Influence Mortality.—

The Importance of the Jaundice Grade.—I have been anxious to find out if there were any factors which might be assessed as having influence on the mortality. It would have been interesting, for example, had one been able to co-relate the pre-operative biochemical findings with mortality. I have already alluded to this relationship in a general way, but the records available did not permit of accurate conclusions. In one instance, however, an interesting point has been demonstrated; it is *the relationship of the jaundice grade to the mortality.* The position has been analysed in 340 cases, and the results are as given in *Table XI.*

Table XI.—Relation of Jaundice Grades to Mortality

Degree of Jaundice	No. of Cases	No. of Deaths	Mortality
			Per cent
Grade I	3	Nil	Nil
„ II	46	6	12·0
„ III	291	129	44·3

The conclusion is obvious: the degree of jaundice has a close association with the mortality, and the explanation seems to be that deep jaundice adumbrates three things—complete duct obstruction, a disorganization of liver function, and delay in adopting measures which might afford relief.

The Causes of Death.—

The Significance of 'Cholæmia'.—This appreciation of the heavy mortality has been a depressing experience. I think all of us knew that it existed, but its recognition was none the less disconcerting. It gave rise to one question: Can it be improved? And, following this line of thought, it is natural that one should inquire as to the exact cause of death. The information obtained in this line of inquiry is given in *Table XII.*

Table XII.—Causes of Death

	Per cent
Post-operative shock	12
Peritonitis	14
Alimentary tract hæmorrhage	12
Cholæmia	58
Chest complications	4

It is apparent, therefore, that a clinical state indicated by the majority of reporters as cholæmia was recognized as the development associated with the fatal issue in the greatest number of cases. The syndrome was described as exhibiting rising temperature, vomiting, loss of consciousness, and anuria.

One cannot but be impressed by the similarity which there is between this sequence of events and the condition described as 'liver death'. Moreover, the evidence provided in the investigation seems to indicate that the principal cause of the mortality in association with the operation for the relief of obstructive jaundice is a failure of metabolic function in certain vital organs, and more particularly in

liver and kidneys. Ivy[4] has made certain statements on the causes of death in obstructive jaundice. He summarizes the position thus :—

1. That as a result of prolonged stasis of bile-salts and pigment there is a disturbance of function in all tissue cells.

2. That resulting changes in metabolism and nutrition are not fatal.

3. That, while bile is lethal in adequate doses, it is not believed that sufficient gets into the blood to prove fatal.

4. That acute liver insufficiency does not occur until *after relief of the obstruction.*

Co-related to this there is a mass of clinical and experimental literature which appears to support the view that an individual can maintain his existence with a complete obstructive jaundice for a remarkably long period of time, but when the obstruction is relieved, and especially if the obstruction is liberated suddenly and completely, a period of danger is incurred. Two features provide the risk: a

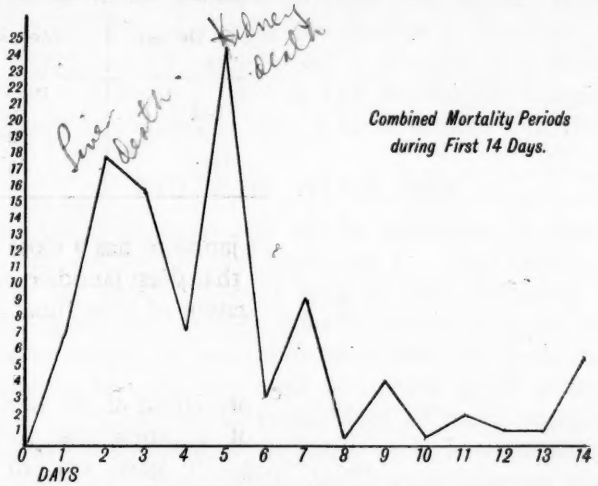

FIG. 274.—Graph to illustrate the time-incidence of death after operation.

failure of liver function with hyaline degeneration and necrosis of the parenchyma ; and some days later (72 to 96 hours) the occurrence of a kidney breakdown, indicated by interstitial hæmorrhage and tubular degeneration. It is suggested that the damaged liver cells release into the circulation some potent toxic substance, which, being excreted by the convoluted tubules of the kidney, results in serious and often fatal damage to the specialized renal epithelium.[5, 6, 7] Such are some of the current views, and the records which have been presented in this investigation appear to support them. I present a graph of the mortality in respect of the days following operation in which death occurred (*Fig.* 274).

You will notice how high is the percentage in the first five days, and, when we appreciate that the so-called cholæmia was indicated as the cause of death in 58 per cent of cases, there seems to be little doubt that a liver-renal death is the actual explanation. I have combined the two terms, but there is a distinction. Liver death develops within the first forty-eight hours succeeding operation, and is characterized by hyperpyrexia, vomiting, hæmatemesis, and coma, while renal death is a later feature, occurring four to five days after operation. Its evidences are listlessness, vomiting, oliguria, the appearance of blood and casts in the urine,

and in fatal cases anuria with coma. These facts have a practical significance, for, if the experimental data have any value, they indicate that the risks are precipitated by the sudden decompression of the biliary tract, and it seems that, if some method could be evolved by which a more gradual relief of pressure were afforded, the immediate results might be improved. Of the various operative measures the two-stage procedure appears to be the one which offers the greatest measure of security, but I imagine that it is not beyond the bounds of possibility for some even more satisfactory method to be evolved.

The Significance of White Bile.—Before leaving the question of mortality I would like to refer to what the investigation has yielded regarding the incidence and significance of white bile. This finding was recorded on 47 occasions; not a high incidence—it works out at 4·4 per cent—but it is the associated mortality which is so disconcerting. Of the 47 cases 38 succumbed within a week of the decompression being carried out, implying a mortality-rate of 82·5 per cent, and, whatever may be the exact explanation of white bile, whether it is an evidence of sepsis and the result of a removal of bile-pigment by leucocytes, or whether it is an evidence of hepatic suppression and in itself a product of the cells which line the bile-ducts, it is in a clinical sense a most ominous and significant sign.

THE LATE RESULTS

It may be that an estimation of the late results was the sole object of the Council in their original remit. I appreciate that, if such was the case, I have gone far beyond their intentions, but at least I have fulfilled the exact terms of the request by tracing the after-history of such cases as were available. The numbers are not great, but they are sufficiently numerous to permit of an analysis being made and conclusions being drawn. I have distinguished between the malignant and the non-malignant cases in this matter of survival.

Malignant Disease.—I have arranged the reports in the form of a table which presents the essential facts (*Table XIII*).

Table XIII.—Late Results of the Treatment of Obstructive Jaundice in Malignant Disease of the Pancreas

Total cases available ..	189
Untraced	49
Number available for analysis	140

Total Cases	Survival Periods	No. of Cases	Percentage
140	Less than 6 months	92	66
	6 months to 1 year	34	24
	1 year to 2 years	5	3
	Over two years	9	7

These figures require no elaboration; it is sufficient to say that 90 per cent of the cases which survive operation are dead within a year. Of the number which survive more than two years the great majority were stated to be enjoying good health, and I think we may take it that this figure represents one aspect of the error in operation diagnosis.

It is a sad picture; it would be difficult to conceive of anything more depressing, and at first sight it seems as though the four reporters who indicated their objection to operation had their case proved for them by these statistics. There is, however, another side to the question, and it is the only bright spot in a field of almost unrelieved gloom; it concerns the condition of the patients during their survival period. Here are the records as far as I have been able to read them (*Table XIV*).

Table XIV.—Condition of Patients during Survival Period (Malignant Disease)

	Per cent
Relief of pain	76
Relief of jaundice (partial or complete)	89
Relief of pruritus	100

This is an encouraging record, and I believe that in itself it may be accepted as sufficient justification for operation.

Chronic Pancreatitis.—I confess that to me the real interest of the investigation lay in this question: What is the fate of the patient who is operated on for obstructive jaundice arising secondary to chronic pancreatitis? In personal conversations I have heard various views expressed, and on the whole they were optimistic, but the literature on the subject is curiously sparse, and it is evident that few attempts have been made to establish accurate knowledge based on the observation of a sufficient number of cases over a long period. For these reasons the present opportunity was welcome. *Table XV* summarizes the position.

Table XV.—The End-results in Obstructive Jaundice arising in Chronic Pancreatitis

Total cases available ..	179
Untraced	7
Number available for analysis	172

Total Cases	Survival Periods	No. of Cases	Percentage
172	Died within 1 year of operation	37	16·3
	Surviving at time of inquiry (1 to 10 years)	135	59·5

I find it difficult to understand why 16·3 per cent of the operation survivals should have succumbed within a year: there appears to be no adequate reason if the original diagnosis was correct. Unfortunately there are insufficient post-mortem records to permit of an accurate answer to the question, but such information as has been available hints that the majority of so-called simple pancreatitis patients succumbing within the year were actually examples of malignant disease the morbid anatomy of which was misjudged at the time of operation. It seems, therefore, that the error in operation diagnosis is as follows: 7 per cent of simple cases are mistaken for malignant disease; while 16 per cent of malignant cases are mistaken for simple disease.

Such being the survival rates, it was important to ascertain the health condition of those who survived. This is essential information, for by it the operative procedures which it is customary to adopt will be justified or condemned. Here is the record so far as I have been able to assess it (*Table XVI*).

Table XVI.—CONDITION OF PATIENTS DURING SURVIVAL PERIOD (SIMPLE CHRONIC PANCREATITIS)

Clinical State	*Per cent*
Enjoying good health	63
Subject to dyspeptic symptoms	28
Recurrence of jaundice	4
Subsequently operated on for biliary disease ..	6

I feel that these are encouraging records, and when it can be claimed that 63 per cent of the patients enjoy good health with complete and permanent disappearance of jaundice our present attitude in respect of operation is vindicated.

I would like, however, to make one comment in respect of the last two groups of figures—patients who developed jaundice subsequently or who were operated on at a later date for biliary disease. You will recall that in describing the operation performed for obstructive jaundice arising in association with simple pancreatitis two types were alluded to, internal drainage or anastomosis and surface drainage. There were 144 of the former and 53 of the latter. Now, the significant point emerges that *patients who experienced recurrence of jaundice or who were operated on at a later date were almost without exception examples of the surface drainage type of operation.* It seems clear that these later complications did not arise subsequent to the anastomosis procedures. I cannot bring forward any evidence to explain the association, but it is my impression that the complication had an infective basis, which is encouraged and possibly initiated by prolonged surface drainage.

CONCLUSIONS

I have appreciated the opportunity which as been afforded me, and, while I am conscious that there are many omissions in the presentation, I have done what I can to bring forward a considered and, I hope, a reasonable survey of the position. As is customary, I offer certain conclusions which I believe to be justified as the result of the investigation.

1. One thousand and thirty-five cases of obstructive jaundice arising in relation to pancreatic disease form the basis of the review.

2. The condition is more common in males than in females in a proportion of 2 to 1.

3. The 'peak' age-incidence (the fifth decade) is common to both simple and malignant conditions.

4. Even at operation the true nature of the morbid anatomy may be difficult to establish. It appears that 7 per cent of simple cases are mistaken for malignant disease, while 16 per cent of the malignant cases are classified as simple.

5. The degree of jaundice may offer a clue to the diagnosis of the malignant case.

6. An immediate direct van den Bergh reaction is an infallible indication of the obstructive nature of the jaundice.

7. The pancreatic source of the obstruction may be established by: (*a*) The sequence of the case history; (*b*) The characters of the pain; (*c*) The state of the gall-bladder.

8. Barium-meal investigation is of little or no value.

9. A knowledge of the degree of the obstruction is afforded by the icteric index and serum-bilirubin readings. The results of these tests aid in assessing the prognosis.

10. An estimate of the extent, the site, and the nature of the pancreatic disease may be arrived at by examination of the fat content of the stool.

11. Sugar-tolerance and blood-urea readings offer reliable information regarding the effects of the obstruction on the liver and renal functions.

12. Blood-coagulation readings yield uncertain results, and the value of the investigation is doubtful.

13. There is diversity of opinion in respect of operating for obstructive jaundice arising from pancreatic disease, but the overwhelming majority favour operation.

14. Of the various types of operation internal drainage or anastomosis procedures are those most favoured, and cholecyst-gastrostomy is the choice in 45 per cent of instances.

15. The operation mortality figures are: (*a*) For all classes of cases, 40·1 per cent; (*b*) In malignant disease of the pancreas, 51·84 per cent; (*c*) In chronic pancreatitis, 19·8 per cent.

16. There is an association between the jaundice grade and the mortality-rate.

17. Cholæmia is quoted as the commonest cause of death. Its effects are exerted about the fifth day, and its development appears to be related to the sudden decompression of the biliary system.

18. 'White bile' is a most ominous finding; the associated mortality is 82·5 per cent.

19. A study of the survival period following operation for malignant disease of the pancreas shows that 90 per cent die within the first year, but during life the large percentage enjoy freedom from pain, jaundice, and pruritus.

20. A similar analysis in chronic pancreatitis shows that 60 per cent of cases are alive at varying periods exceeding one year after operation, and are enjoying relatively good health. When relapses occur they are apt to be associated with previous operations of the surface-drainage type.

The foregoing conclusions have various points of interest, but two in particular appear to support the claim that operation is the proper course to adopt in cases of obstructive jaundice attributable to pancreatic disease. These two conclusions are: (1) The difficulties in diagnosis, so that the simple case may be mistaken for a malignant one; and (2) The freedom from symptoms which is enjoyed by the survivors—a temporary relief in the malignant case, and one that is more lasting in chronic pancreatitis.

There is one obvious question: Has the investigation revealed means by which the mortality figures can be improved? The review seems to offer two suggestions: (1) Operate early, before liver and renal functions are seriously impaired; and (2) When operating on cases which are profoundly jaundiced adopt a two-stage method, for by this means the risks of a sudden biliary decompression are diminished.

REFERENCES

[1] COUGHLIN, W. T., and MCCAUGHAN, J. M., *Jour. Missouri State Med. Assoc.*, 1935, xxxii, 425.

[2] MCGOWAN, J. M., BUTSCH, W. L., and WALTERS, W., *Jour. Amer. Med. Assoc.*, 1936, June 27, 2227.

[3] MOYNIHAN, LORD, *Abdominal Operations*, 4th ed., 1926, 529.

[4] IVY, A. C., *Jour. Amer. Med. Assoc.*, 1930, xcv, Oct. 11, 1068.

[5] BOYCE, F. F., and MCFETRIDGE, E. M., *Arch. of Surg.*, 1935, xxxi, 105.

[6] STEWART, H. L., and LIEBER, M. M., *Arch. of Pathol.*, 1934, xviii, 30.

[7] CONNEL, F. G., *Ann. of Surg.*, 1934, c, 319.

VISITS TO SURGICAL CLINICS AT HOME AND ABROAD

ALTOUNYAN OF ALEPPO

IN 1936 a party of Hellenic Travellers were making a journey by automobile from Alexandretta, in the North of Syria, across the desert to Baghdad. As the journey is long and strenuous, some anxiety was felt lest certain members of the party who were no longer young might fall ill and need medical attention. So it was considered desirable to find out what accommodation could be procured should the need arise. Reference to the Medical Directory revealed the fact that there is a hospital at Aleppo called the Altounyan Hospital, so a visit was paid to it to see what it was like.

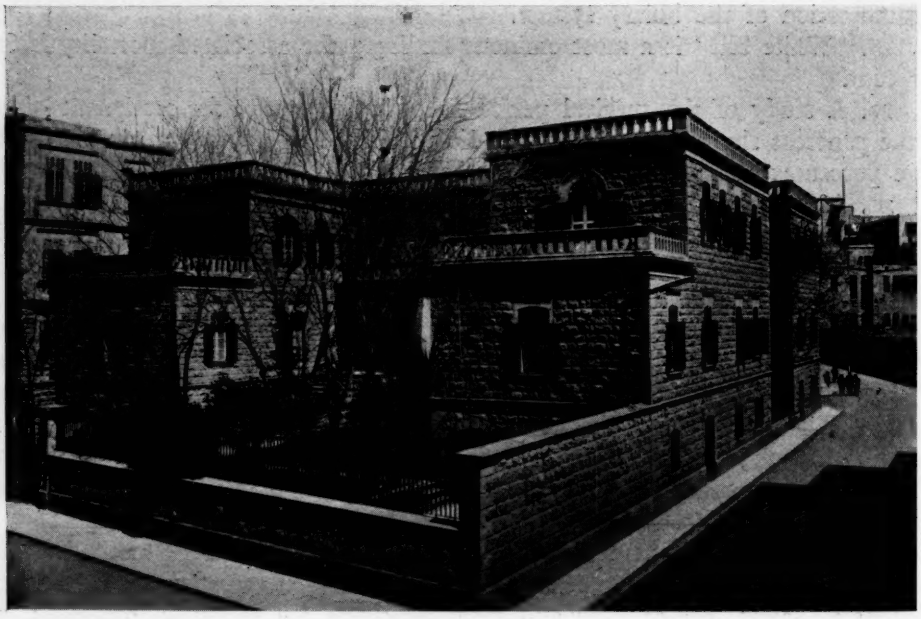

FIG. 275.—The Altounyan Hospital.

Aleppo is a very ancient city where Hittites, Assyrians, Egyptians, and Crusaders, all in their turn, have fought and perished. From the sixteenth century the city has been in the hands of the Turks, until October 26, 1918, when it was occupied by the Fifth Cavalry Division of the Egyptian Expeditionary Force and came under the British control. Then in 1920 it was placed under the French mandate.

The town, with its magnificent citadel, lies on the edge of the desert which separates Syria and Palestine from Mesopotamia. It is largely eastern in character,

which makes it all the more surprising to find there a modern hospital—for the Altounyan is modern in all respects. It is a private hospital of about thirty beds, and in a small way it reminds one of the Mayo Institute at Rochester, for there is the same attention to organization and detail to be seen at the two places. (*Figs.* 275–278.)

It is rather astonishing to a self-satisfied westerner to find on the edge of the desert full facilities for the examination of the blood, a complicated chemical analysis, or an endoscopic and fluoroscopic examination of the urinary tract.

The operating theatre is admirable and lavishly equipped with instruments of all sorts and for every type of operation. An operation book has been kept since

FIG. 276.—One of the wards.

1891, and in 1935 showed that 15,868 operations had been performed. These operations were by no means of a minor character, but were representative of all branches of surgery and gynæcology.

The nursing is in the charge of an English matron, Miss Still, and she has a training school of some twenty Armenian girls, so that the ideals of western nursing are being spread in Syria.

The creation and maintenance of this medical organization is the work of one man, and perhaps the readers of the BRITISH JOURNAL OF SURGERY will be interested to read the story of his life.

Asadour Aram Altounyan (*Fig.* 279) was born in 1857 at Sivas, a town in Asia Minor, about 100 miles south of the Black Sea. He was the son of Christian Armenian parents, both his father and uncle being "empirical doctors"—that is to say, medical practitioners without a medical degree, though not necessarily without medical training.

At the age of 7 Altounyan went to the National school, where instruction was rudimentary, only reading and writing being taught. Here he remained till the age of 14, when he started work in the town of Sivas with an American doctor, Dr. West, who was teaching boys medicine in his dispensary. His pupils were as a rule badly educated, hardly able to read or write; yet many of them possessed considerable ability, and so, with the practical experience gained, became useful doctors.

The teaching given by Dr. West was not elaborate. He taught anatomy, mostly didactically, in bad Turkish and with the aid of books. Any dissection that was done was by stealth. Altounyan relates that once after the death of a pauper who had no relations, the pupils resurrected the corpse at night, took it to a secret place, and dissected it for several weeks. After the anatomy there was a short

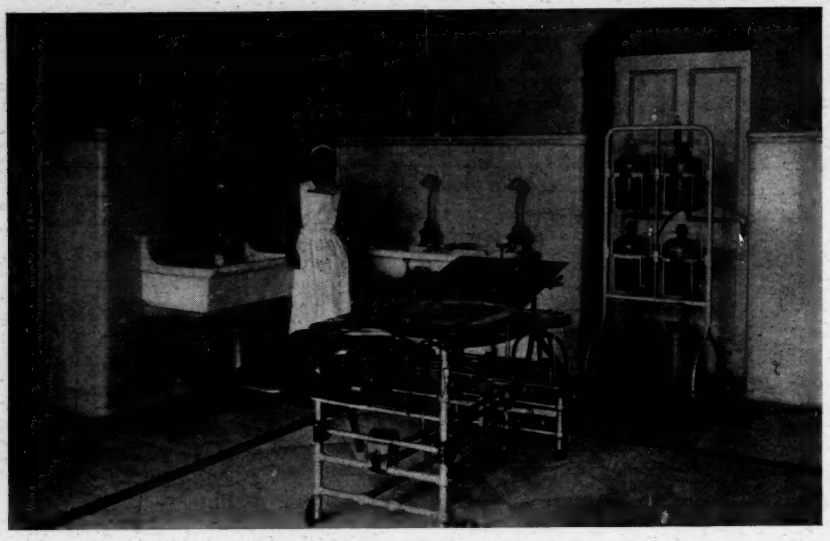

FIG. 277.—The operating theatre.

course in chemistry and materia medica, followed by instruction in medicine and surgery. At the end of the course a certificate of proficiency was given, after which some of the pupils would go to Constantinople to take the Turkish certificate of Santé and others drifted into medical practice.

During this period Altounyan was miserably poor and without money to buy books. He says that he worked a hundred hours, at threepence an hour, in order to buy a book on anatomy. This shows the sort of stuff he was made of.

Meanwhile Altounyan's uncle and cousin were practising medicine at Amasya, a town not far from Sivas, and Altounyan went there to work for them, making up medicines, washing bottles, looking after the horses, and making himself generally useful.

Then, when about the age of 18, something happened which changed the whole course of his life and started him on the career from which he never looked back. One of the professors at the school of Aintab married Altounyan's cousin and he got the chance to go to Aintab to live with them and to study medicine.

The city of Aintab is in Asia Minor, about fifty miles north of Aleppo. About the year 1873 the American Congregationalists sent a mission to Turkey and established a school at Aintab called the Central Turkey College, with T. C. Trowbridge as its president. Attached to this College was a medical school, and here Altounyan got the chance of learning western medicine and he received the diploma given by the College in 1881. The authorities of the College, recognizing the promising qualities of their pupil, provided him with money to go to America to pursue his studies. He entered the Columbia College, New York, and in 1885 took the Diplomas of the College of Physicians and Surgeons of New York. He came out at the top of his class, securing for himself the chance of an internship at the Roosevelt Hospital under Sands. As this appointment did not seem to Altounyan

FIG. 278.—The out-patient clinic.

to offer the sort of experience he wanted, he sold the appointment to the second man, securing 100 dollars on the deal, and used the money to visit the medical centres of Chicago and Ann Arbor, and also to take a course of microscopical anatomy and pathology in a laboratory at New York.

From America, Altounyan journeyed to Heidelberg, where he learnt German really well. He spent a term in Erb's Clinic and also went to Berlin, where, after waiting three months, he secured a place in Koch's laboratory and took a full course in pathology.

Having now completed a good course of education and a very unusual one for an Armenian, Altounyan returned to Constantinople to secure permission to practise in Turkey, and in 1887 he secured the diploma from the École Impériale de Médecine de l'Empire Ottoman.

Thus brilliantly qualified, he returned to Aintab as a professor, to repay in kind the benefits he had so freely received as a student. He taught pathology,

medicine, and practical surgery, being associated with two other professors, Dr. Shepherd, who came from the States, and Dr. Graham, from Canada.

The pay was not high, the professors getting £120 per annum for whole-time work, any earnings going to the College.

This combination did not last long, for in 1889 the Aintab medical school came to an end, owing to the Turks refusing any longer to recognize its Diploma. Dr. Shepherd remained in practice in Aintab, Dr. Graham moved to Beirut, while Altounyan after some journeying settled in Aleppo.

In 1887 Altounyan married Miss Harriet Riddell, of Armagh, the sister of Canon Riddell, who was then matron of the Aintab Hospital.

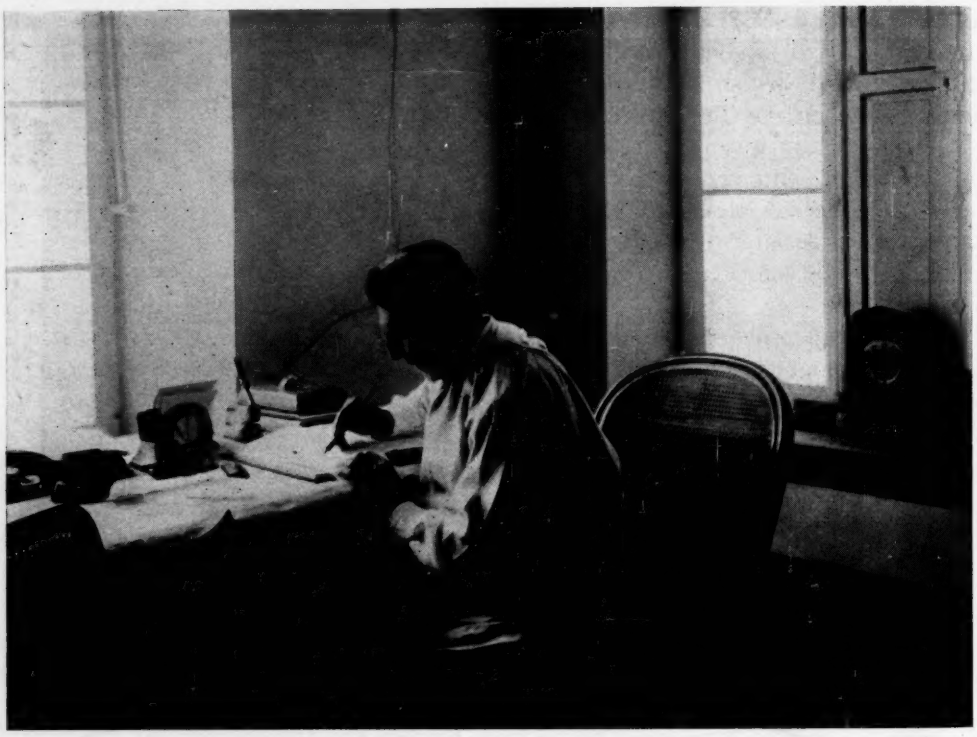

FIG. 279.—Photograph of Dr. A. A. Altounyan, the creator of the hospital.

After the closure of the hospital Altounyan felt the need of making money, so he made a tour of the towns of Asia Minor, settling for a week or a month, seeing and treating patients, and then moving on. By this means he gained enough to visit Vienna, where he worked under Billroth and von Eiselberg and also to come to London so that his wife might have her baby in England. His son Ernest was born in Paddington, while the father, always working, took a three months' course at St. Mary's Hospital.

After this he returned to Aleppo and started practice, doing all sorts of work—medicine, midwifery, tropical diseases, and surgery. As regards surgery, Aleppo was then in the dark ages, and he introduced into that city western medicine and aseptic surgery. At first most of his operations were done in a single room, painted

white, with two samovars to provide hot water. Most of the patients were taken away by their friends and nursed at home, but he had one room in his house which could be used for very ill patients.

He kept abreast of the times by reading current literature, mostly German and English, and every second year he would visit a European centre such as Vienna, Berlin, or Berne, and so he came to know the best surgeons in Europe.

Then in 1908 he built his own hospital, paying on Saturday with money he had earned during the week.

Early in his stay at Aleppo he gained the confidence of the Turks by saving the life of the child of a high Turkish official. The child was desperately ill with high fever and hæmoptysis and was despaired of by the doctors. Altounyan recognized the disease as typhoid fever and the child recovered. This confidence gained stood him in good stead, for during the Great War he remained unmolested, and with three British nurses looked after the families of Turkish officers, and then later, when the Turks retreated, he looked after the British wounded.

Though now advanced in years, Altounyan is still active in mind and body, and he related with vivacity the story of his life in his study full of rare Persian rugs, where he was pleased to show one which T. E. Lawrence, a constant visitor to the house, declared to be worth the sack of Aleppo. He is now happy in the assistance of his son, Ernest Altounyan, M.C., M.D. Cantab., whom he has trained to follow him.

Like many other surgeons Altounyan has not written much. His influence has been through his personal qualities, and by these he is known and respected and will be long remembered throughout the Near East by his sobriquet of the "Hakim Amerikani".

EXPERIMENTAL SURGERY

THE BEHAVIOUR OF THE BLOOD-VOLUME IN INTESTINAL OBSTRUCTION AND STRANGULATION*

By IAN AIRD

All forms of intestinal obstruction present three features which tend to induce a reduction in the volume of effective circulating blood :—

1. The capillaries and veins of the distended bowel above an occlusion are congested, and the blood which fills these distended vessels is no longer available for the needs of the general circulation. This is the ' splanchnic congestion ' which has been advanced as the cause of death in intestinal obstruction. In strangulation splanchnic congestion is extreme, and from the engorged vessels of the affected bowel segment considerable quantities of blood may pass into the lumen and into the peritoneal cavity. The blood lost in this way into the vessels, the lumen of the intestine, and into the peritoneal cavity, must tend to reduce the volume of whole blood, and to affect the volume both of cells and of plasma.

2. In simple occlusion of the intestine, plasma passes from congested vessels into the tissues of the bowel wall, rendering them œdematous, and into the lumen to increase the volume of content. One would expect this loss of plasma to tend to reduce the plasma-volume, and so the total blood-volume, but to have little effect on the cell-volume.

3. The congested bowel in obstruction or strangulation is no longer capable of absorbing water and salts ; indeed, in high occlusion, fluid and electrolytes fail to reach the absorptive mucosa of the lower bowel. Dehydration and demineralization follow in the organism, and the higher the obstruction, the more rapid is their development. In high obstruction, dehydration and chloride depletion are alone responsible for death, since death can be indefinitely postponed by the administration of saline solutions intravenously or into the lower unobstructed bowel. Dehydration, however, is not necessarily associated at first with a reduction in blood-volume. For a time, plasma passes from the tissue spaces of the body to the blood, and replaces the fluid which has been lost in the digestive juices. Before death, however, the blood becomes highly concentrated and viscid, the corpuscles are closely packed, and the red-cell count is elevated[9]—effects which suggest that in high obstruction there is a terminal reduction in plasma-volume, and presumably a consequent decrease in blood-volume, the cell-volume being relatively unaffected.

Measurement of the loss of blood and plasma from the general circulation into the distended vessels, tissue spaces, and lumen of obstructed or strangulated bowel, and into the peritoneal cavity, has been attempted by certain indirect methods. Foster and Hausler,[8] to measure the blood-loss in strangulation, surrounded a strangulated bowel loop by a rubber bag, and after death compared the weight of

* From the Surgery Research Department, University of Edinburgh.

the contents of the bag with the weight of a loop of bowel similar in length to the strangulated loop. This method, in the hands of Foster and Hausler and of later workers,[1, 3, 4, 10] indicated that in strangulation of long bowel loops enough blood is lost from the congested vessels to cause death by a form of internal hæmorrhage. It has, however, been argued that the introduction of a rubber bag, which prevents peritoneal reabsorption of the fluid lost from the strangulated loop, exaggerates the apparent blood-loss. Knight and Slome,[11] and Scott and Wangensteen,[13] preferred to measure the blood lost into the bowel wall and lumen by comparing the weight of strangulated bowel with the weight of a similar length of normal bowel; the blood lost into the peritoneal cavity they measured by mopping that cavity dry with absorbent wool, and by comparing the weight of dry wool used with the weight of the same wool sodden with peritoneal fluid. Knight and Slome found that the volume of lost blood was smaller when estimated in this way than when measured by the rubber bag method, but Scott and Wangensteen's figures correspond fairly closely with those of the advocates of the rubber bag.

The main objection, however, to all indirect methods of estimating splanchnic congestion is the difficulty of obtaining for comparison two bowel loops of equal length. A slight difference in tension of the two loops when their lengths are measured may lead to a considerable error in the estimate of blood-loss.

Indirect methods of measuring the reduction of blood-volume have given such varied results, and are open to so much criticism, that it seemed essential to examine this important aspect of intestinal obstruction finally by direct estimation of the blood-volume. E. Derra[7] has measured the blood-volume of animals during the first few days after the establishment of intestinal obstruction. Only one or two post-operative estimations were made, but these showed sometimes a fall of from 13 to 29 per cent of the total blood-volume.

It was decided, by performing estimations at short intervals, to obtain curves of the blood-volume before and after the establishment of various forms of experimental intestinal obstruction. In each of a series of nineteen dogs, the normal blood-volume was first determined. Numerous estimations of the blood-volume were made until five successive readings varied less than 10 per cent from their mean; these five readings were taken as a base line. Acute intestinal obstruction was then induced, and the blood-volume estimations were continued at daily or even more frequent intervals until death.

METHOD OF MEASUREMENT OF THE BLOOD-VOLUME

Of the many techniques devised for measurement of the blood-volume, four are still fairly commonly used. Of these, direct measurement of the blood removed from the body and minced tissues of the animal after death gives only a single measurement, and offers no record of changes in the blood-volume during life.

A second method, devised by Haldane, entails the administration of a known quantity of carbon monoxide by inhalation, and the subsequent colorimetric measurement of carbohæmoglobin per cubic centimetre of blood.

For two reasons the carbon monoxide technique was avoided. In the first place, it is difficult to persuade animals to inhale an oxygen–carbon-monoxide mixture from a bag, and, secondly, when the red-cell volume is already reduced by disease (in extensive strangulation, for example) the carbon monoxide inhaled

reduces the already impaired oxygen-carrying capacity of the blood, and may hazard the life of the animal.

J. D. Robertson,[12] in a study of the effect of hæmorrhage, has used hæmoglobinometry as an index of the changes in blood-volume. This method is applicable only in cases where the volume of blood removed from the circulation is accurately known.

The dye method of Brown and Rowntree[6] was finally adopted, as essentially simple, sufficiently accurate for the present purpose, and, when modified in certain of its details, suitable for repetition at short intervals. The dye used was vital red. To oxalated plasma obtained before the injection of dye was added a known quantity of vital red. This furnished a new standard solution on each occasion for colorimetric comparison with oxalated plasma obtained after the intravenous injection into the animal of a known quantity of the same dye and suitably diluted. In this way the plasma-volume was estimated, and the blood-volume and cell-volume were obtained from the plasma-volume by the use of the hæmatocrit.

The main disadvantage of the dye method is that it takes an appreciable time for the dye to become equally diffused throughout the body, yet dye begins to leave the circulation within a few minutes of injection. It is difficult therefore to choose a moment when the dye shall have been generally diffused, yet shall not at that time have been lost in any quantity from the circulation. Three separate specimens of blood were therefore removed for colorimetry at intervals of three, five, and eight minutes after injection of the dye, and the most densely stained of these was used for colorimetric comparison. On almost every occasion the second specimen was the most densely stained; at three minutes diffusion was hardly complete, and at eight minutes excretion had already begun.

In making repeated estimations of the blood-volume by the dye method, dye remains to some extent unexcreted, and accumulates in the plasma. The plasma may then be already dye-stained before any new dye is injected. As Brown and Rowntree have shown, this accumulation of dye is unimportant, since a new colorimetric standard is obtained on each occasion, and the residual dye therefore stains equally the standard solution and the unknown.

The exact details of the method are these: Into a syringe containing 1 c.c. of 1·6 per cent sodium oxalate solution is drawn precisely 5 c.c. of blood (the 'first specimen') from one saphenous vein, and into that vein is then injected a 1·5 per cent solution of vital red. The amount introduced varies with the size of the animal. One c.c. of vital red solution is injected per 4 kilos of body-weight, or approximately 1 c.c. per 200 c.c. plasma, if the plasma-volume is taken as 50 c.c. per kilo body-weight. After intervals of three, five, and eight minutes, three specimens of blood, each precisely 5 c.c., are then similarly withdrawn from the contralateral saphenous vein by a fresh needle, and the most densely stained of these is used for colorimetric comparison.

The first and second specimens, each of 5 c.c. of blood and 1 c.c. of oxalate solution, are then centrifuged in graduated tubes at a standard rate of 3500 r.p.m. for a period of half an hour, and the percentage of plasma is measured. To 2 c.c. of oxalated plasma from the first specimen is then added 2 c.c. of a 1–200 solution of 1·5 per cent vital red and 4 c.c. of physiological saline. The standard coloured mixture thus obtained is compared colorimetrically with a mixture of

2 c.c. of the stained plasma of the second specimen of blood and 6 c.c. of physiological saline. If the plasma-volume is, as anticipated, 50 c.c. per kilo body-weight, the tints of the two specimens are identical, provided that allowance is made for oxalate dilution of the plasma. The plasma volume is obtained by the formula :—

$$\text{Plasma-volume} = \frac{200\ AD}{C},$$

where A is the volume of dye injected;

D is the dilution factor $\left(\frac{\text{vol. of plasma in 5 c.c. blood}}{1 + \text{vol. of plasma in 5 c.c. blood}}\right)$ which makes allowance for oxalate dilution;

C is the concentration of dye in the second specimen, the concentration in the standard being taken as unity.

EXPERIMENTAL RESULTS

Studies of the blood-volume, plasma-volume, and cell-volume were performed before and after the induction of intestinal obstruction and strangulation. The experiments are considered below in three categories, according to the variety of experimental obstruction established.

1. High Small-intestine Occlusion.—After division and inversion of the lower duodenum or upper jejunum, the dogs lived for several days. There was first little alteration in the blood-volume, but before death a reduction of from 14 to 44 per cent occurred. In this form of occlusion the cells suffered only a slight loss, and the fall in blood-volume was due to a diminution in the volume of the plasma (*Table I* and *Fig.* 280).

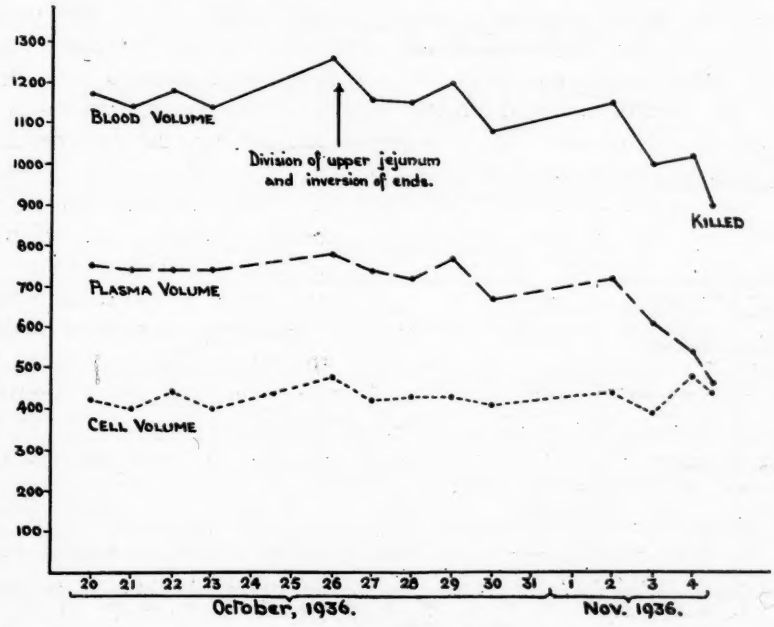

FIG. 280.—*Dog* 5. Weight 16·30 kilos. High small-intestine occlusion. Depletion mainly of plasma-volume.

Table I.—HIGH SMALL-INTESTINE OCCLUSION.

No. of Expt.	Level of Occlusion	Duration of Life	Mean Blood-volume	Reduction in Blood-volume	Reduction in Plasma-volume	Reduction in Cell-volume
			c.c.	per cent	per cent	per cent
1	Terminal duodenum	5 d.	1030	25	43	–
2	Below ampulla	11 d.	1160	14	33	–
3	Below ampulla	6 d.	1360	23	44	7
4	Terminal duodenum	13 d.	1400	30	47	–
5	Terminal duodenum	9 d.	1180	44	32	–
6	Below ampulla	17 d.	1400	18	31	–

2. Low Small-intestine Occlusion.—Division of the lower ileum, with ligation and inversion of the open ends, had a variable effect upon the blood-volume. In *Dogs* 7 and 10, no significant change occurred in the blood-volume, in the plasma-volume, or in the cell-volume so long as life lasted. In both these animals the small intestine at the time of death was moderately dilated and its walls slightly cyanosed and œdematous. In *Dogs* 8 and 9 there was a considerable reduction in plasma-volume, in blood-volume, and in cell-volume, the plasma suffering to a greater degree, however, than the cells. In these two animals there was gross dilatation and venous congestion of the whole small intestine at the time of death. (*Table II* and *Figs.* 281, 282.)

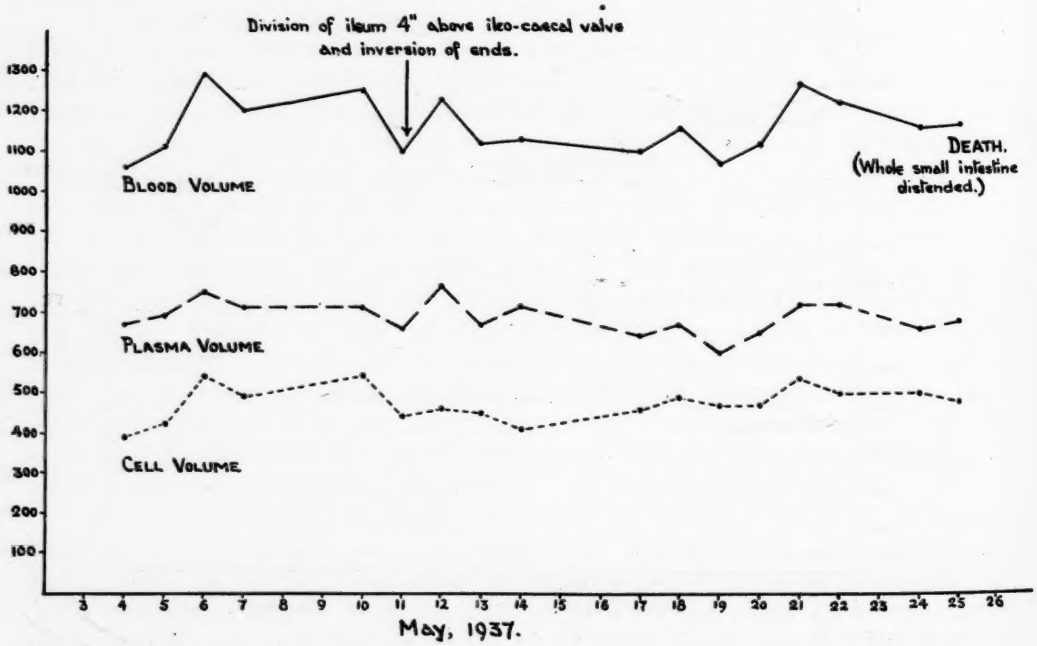

FIG. 281.—*Dog* 7. Weight 14·3 kilos. Low small-intestine occlusion. No alteration in blood-volume.

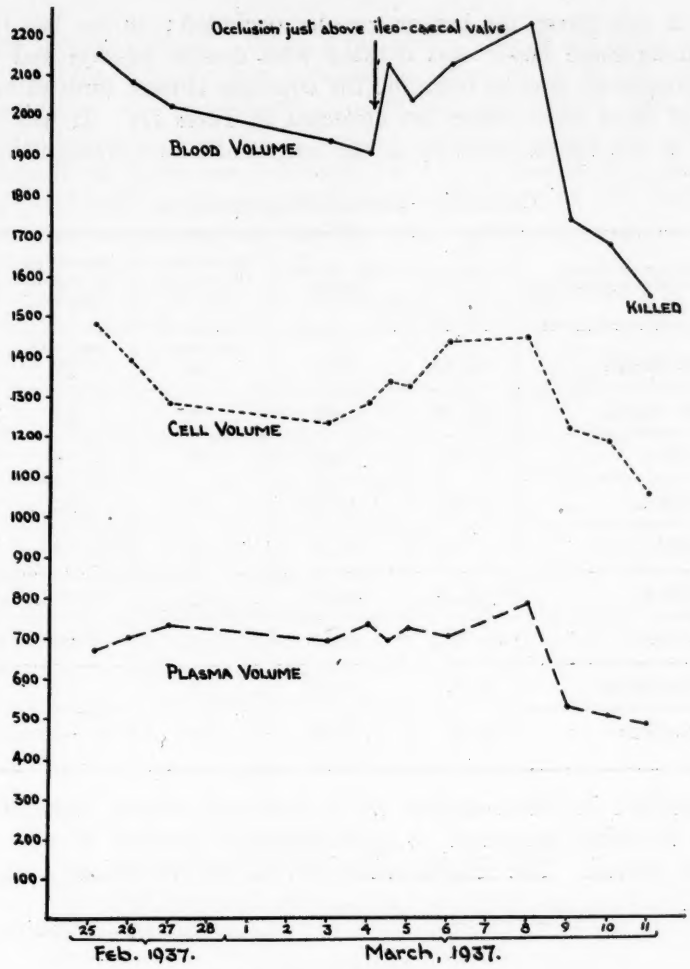

FIG. 282.—*Dog* 8. Weight 21·5 kilos. Low small-intestine occlusion. Depletion of blood-volume at death, associated with gross congestion of the distended bowel.

Table II.—LOW SMALL-INTESTINE OCCLUSION

No. of Expt.	Level of Occlusion	Duration of Life	Mean Blood-volume	Reduction in Blood-volume	Reduction in Plasma-volume	Reduction in Cell-volume
			c.c.	per cent	per cent	per cent
7	Lower ileum	14 d.	1170	—	—	—
8	Lower ileum	7 d.	2020	23	31	20
9	Lower ileum	9 d.	1610	44	50	37
10	Lower ileum	9 d.	1410	—	—	—

3. Complete Venous Strangulation.—In nine animals, venous strangulation was induced by the ligation of all veins draining a bowel loop. In the first six

experiments in this group the lumen was not occluded; in the last three experiments the strangulated bowel was divided with double ligation and invagination as a closed, congested loop to resemble the common clinical form of strangulation. The results of these experiments are arranged in *Table III*. It will be seen that the duration of life varied inversely as the length of bowel strangulated.

Table III.—VENOUS STRANGULATION

No. of Expt.	Amount of Small Intestine Strangulated	Duration of Life	Mean Blood-volume	Reduction in Blood-volume	Reduction in Plasma-volume	Reduction in Cell-volume
			c.c.	per cent	per cent	per cent
11	Whole length	7¼ hr.	870	44	37	47
12	Whole length	2½ hr.	430	52	50	59
13	One-third	12 hr.	740	30	16	50
14	One-third	5 hr.	1170	49	30	66
15	One-fifth	5 hr.	1240	40	30	55
16	One-third	5½ hr.	1480	43	27	68
17	One-tenth	24 hr.	1060	17	30	8
18	One-twentieth	4 d.	2150	15	21	10
19	One-fortieth	12 d.	1570	—	—	—

The reduction in blood-volume which followed venous strangulation of the whole small intestine amounted to approximately one-half of the total blood-volume of the animal. The reduction affected the cell-volume to a slightly greater

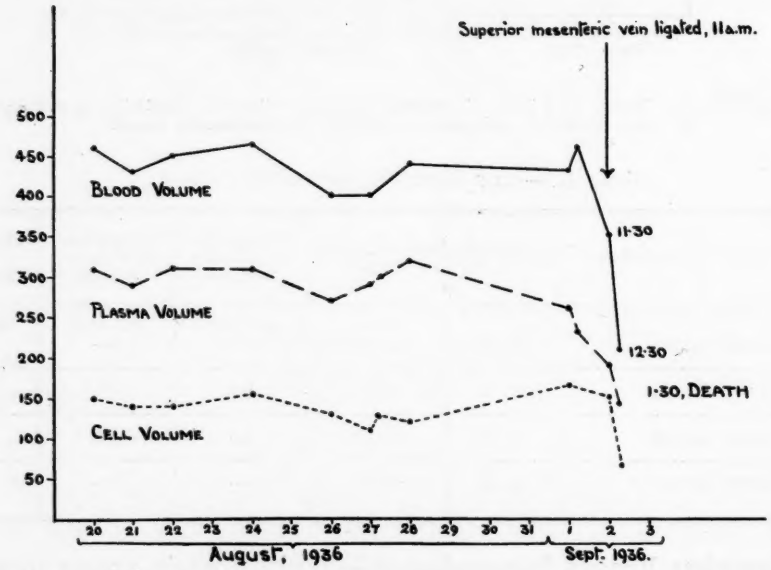

FIG. 283.—*Dog* 12. Weight 6·8 kilos. Massive venous strangulation. Depletion of volume of whole blood.

extent than the plasma-volume. When a third or a fifth of the small intestine was strangulated, the blood-volume was reduced by from 30 to 50 per cent; in this type of strangulation the cell-volume was grossly diminished, and the plasma-volume was relatively little affected. In the last three experiments, after strangulation of a short bowel loop, life continued for a considerable time (in *Dog* 19 for twelve days), the blood-volume was not greatly altered, and the plasma suffered a greater reduction in volume than did the cells. (*Figs.* 283, 284.)

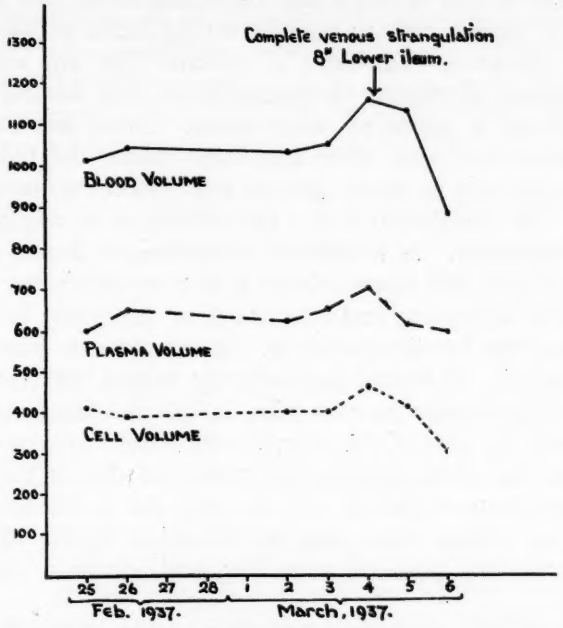

FIG. 284.—*Dog* 17. Weight 17·5 kilos. Strangulation of 8-in. loop of ileum. Slight reduction of blood-volume.

DISCUSSION

The changes in blood-volume which follow the induction of high small-intestine occlusion are easily explained by dehydration. In high occlusion, the digestive juices cannot pass the level of obstruction to be reabsorbed lower in the bowel, and the plasma-volume tends to fall. For a time tissue fluids replace the lost plasma, and at first there is no reduction in blood-volume. Before death, however, the dehydrated tissues can no longer replace the fluid elements of the blood; the plasma-volume falls, and with it the blood-volume, but the cell-volume is unaffected.

In low small-intestine obstruction, death occurred in two animals without any reduction in blood-volume, plasma-volume, or cell-volume. Blood-loss and severe dehydration are thus not necessarily concerned in the fatal issue in this variety of obstruction. In the other two animals there was at death a considerable diminution in the plasma-volume, indicative of a measure of dehydration, and a reduction also in the cells. The cell reduction is doubtless due to the gross intestinal congestion which in *Dogs* 8 and 9 was a striking feature at autopsy. Even in these two animals the reduction in blood-volume was not sufficient in itself to account for their deaths, and reduction in blood-volume cannot be considered an essential lethal factor in low small-intestine occlusion.

Massive venous strangulation of one-fifth or more of the small intestine is followed by a striking fall in the cell-volume to one-half or less of its normal level, and the blood-volume also falls to a slightly less extent. Such a loss of effective circulating blood is comparable to the blood-loss in a fatal external hæmorrhage, for Blalock[5] has shown that the loss by hæmorrhage of one-half of the blood-volume is likely to lead to death.

It is perhaps unjustifiable immediately to conclude that the profound fall in blood-volume is due to loss of blood into the strangulated loop and into the peritoneal cavity. It is impracticable to establish strangulation without the performance of an abdominal operation. Yet there is evidence that any operative procedure may reduce the volume of effective circulating blood, and that the normal level may not be re-attained for a period of some hours. Derra has observed that after abdominal operations, and even after anæsthesia alone, the red-cell volume may be reduced by 10 per cent or more, and the total blood-volume may in some cases be reduced also. The blood-volume as a rule returns to its normal level in twenty-four hours after operation. It is difficult, therefore, to decide how much of the reduction in cell-volume and blood-volume is due to anæsthesia, how much to the trauma of abdominal operation, and how much to the strangulation itself. These complicating factors can be disregarded in the experiments which concern simple occlusion of the bowel; in simple occlusion the animal survives for several days, and the immediate post-operative reduction in blood-volume (when there is one) has time to recover. In nine of the animals which were subjected to simple occlusion of the lumen, the blood-volume was measured during the first twenty-four hours of the post-operative period. In no case did a fall of more than 10 per cent occur; in one animal there was no alteration in the blood-volume when measured during the first post-operative day, and in three animals a slight rise was recorded.

It seems reasonable, therefore, to assume that the fall in blood-volume consequent upon massive intestinal strangulation, is due less to the 'shock' of laparotomy and anæsthesia, than to the actual state of strangulation. The depletion of blood-volume and cell-volume, moreover, corresponds fairly closely to previous figures obtained by weighing the strangulated bowel and measuring the fluid lost from it. Holt,[10] measuring the contents of a rubber bag in which a long strangulated loop was incarcerated, estimated a blood-loss of more than 50 per cent. Using the same method, I obtained in a previous investigation[3] the figures given in *Table IV*. It will be seen from these figures that when one-half of the small intestine is strangulated, the apparent blood-loss amounts to approximately one-half of the estimated blood-volume of the animal (blood-volume equals approximately one-fifteenth of the body-weight).

Scott and Wangensteen,[13] weighing strangulated loops and the peritoneal transudate from them, also established that in long-loop strangulation, from 34 to 66 per cent of the estimated blood-volume may be lost; the average loss in their series was 55 per cent. Knight and Slome found a lesser degree of blood-loss in their series of long-loop strangulations. Using, in three animals, loops varying in length from 32 to 40 cm., they measured the blood-loss by weighing the strangulated loop, and the peritoneal fluid mopped out with a known weight of absorbent wool; measured in this way, the lost blood varied in amount from 1·5 to 3·7 per cent of the body-weight (or approximately 20 to 49 per cent of the blood-volume).

The difficulty of measuring off a length of bowel equal to the strangulated loop has already been mentioned, and the further difficulty of mopping a dog's peritoneal cavity dry of fluid is sufficiently obvious. Nevertheless, the figures obtained by Holt, by Scott and Wangensteen, and by myself in measuring the fluid loss, correspond so strikingly with the present estimate of blood-volume depletion, that it seems likely that massive strangulation induces a serious reduction of blood-volume by direct loss of blood into the strangulated bowel and into the peritoneal cavity.

Table IV.—Blood-loss in Massive Intestinal Strangulation

No. of Expt.	Fraction of Small Intestine Strangulated	Weight of Blood Lost	Estimated Blood-volume	Blood-loss as Percentage of Blood-volume
1	One-half	110	210	52
2	One-half	80	188	43
3	Two-sevenths	42	120	35
4	One-quarter	39	172	23
5	Two-ninths	42	158	27
6	Two-ninths	53	165	32
7	One-fifth	31	135	23
8	One-fifth	52	188	28
9	One-seventh	33	150	22

Massive strangulation of one-fifth or more of the small intestine, established by operation in the experimental animal, is thus followed by a depletion of the blood-volume and cell-volume comparable with the fatal depletion which accompanies rapid external hæmorrhage. When allowance is made for the inaccuracy of the previous methods of measurement of blood-loss in strangulation, the figures obtained by these methods largely correspond with the figures of blood-volume depletion presented here, and suggest that the reduction in blood-volume is due rather to loss of blood into the strangulated loop and into the peritoneal cavity, than to the effects of anæsthesia and laparotomy.

In strangulation of shorter loops (one-tenth of the small intestine or less) the animal survives for some days, and the blood-volume is relatively little reduced at death. What reduction there is affects the plasma rather than the cells, and the blood-volume curve approximates more closely to its curve in simple occlusion; so far as the effect on the blood-volume is concerned, short-loop strangulation bears more resemblance to simple low small-intestine occlusion than to massive strangulation.

CONCLUSIONS

In high small-intestine occlusion, there is at the time of death a reduction in blood-volume and in plasma-volume due apparently to the dehydration which, with demineralization, is the dominant factor in causing death.

In low small-intestine occlusion death may occur without reduction of the blood-volume. If, however, the animal lives until dilatation and congestion of the obstructed bowel supervene, then the blood-volume may be markedly reduced at the time of death, but not sufficiently reduced to be solely responsible for death. The reduction in blood-volume seems to be due partly to the loss of whole blood into the congested intestinal vessels, and partly to dehydration, since the plasma loss is greater than the cell loss.

In experimental venous strangulation, if the strangulated loop is long, reduction of the blood-volume is considerable, and may be held to be the main cause of death, just as it is the main cause of death in massive external hæmorrhage.

In venous strangulation of a short bowel loop the reduction in blood-volume, while often considerable, is not sufficient in itself to occasion death. The reduction appears to be due to terminal dehydration, the result of concomitant occlusion, rather than to actual blood-loss.

The experiments have been conducted in the Surgery Research Department of the University of Edinburgh. During their performance Sir David Wilkie has been generous in advice and in encouragement. I have frequently enjoyed recourse to Mr. W. C. Wilson's wide experimental knowledge. The colorimetric studies were conducted in the Biochemistry Department of the Royal Infirmary, by courtesy of Professor D. M. Lyon, and with guidance from Dr. Harold Scarborough. During the performance of the experiments, the author has been in receipt of a personal grant from the Medical Research Council. Part of the animal expenses has been borne by the Earl of Moray Endowment Fund of Edinburgh University.

REFERENCES

[1] AIRD, I., *Proc. Soc. Exper. Biol. and Med.*, 1935, xxxii, 1593.
[2] AIRD, I., *Edin. Med. Jour.*, 1936, xliii, 375.
[3] AIRD, I., *Ibid.*, 1937, xliv, 28.
[4] AIRD, I., *Proc. Roy. Soc. Med.*, 1936, xxix, 991.
[5] BLALOCK, A., *Arch. of Surg.*, 1927, xv, 766.
[6] BROWN, G. E., and ROWNTREE, L. G., *The Volume of the Blood and Plasma*, 1929, 219. Philadelphia: W. B. Saunders.
[7] DERRA, E., *Deut. Zeits. f. Chir.*, 1936, ccxlvii, 82, 187.
[8] FOSTER, A. C., and HAUSLER, R. W., *Arch. of Internal Med.*, 1924, xxxiv, 697.
[9] FOSTER, A. C., and HAUSLER, R. W., *Ibid.*, 1925, xxxvi, 31.
[10] HOLT, R. L., *Brit. Jour. Surg.*, 1934, xxi, 582.
[11] KNIGHT, G. C., and SLOME, D., *Ibid.*, 1936, xxiii, 820.
[12] ROBERTSON, J. D., *Jour. of Physiol.*, 1935, lxxxiv, 393.
[13] SCOTT, H. S., and WANGENSTEEN, O. W., *Proc. Soc. Exper. Biol. and Med.*, 1932, xxix, 748.

SARCOMA OF THE INTESTINE IN CHILDREN

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A boy was recently operated on at the Hospital for Sick Children for a chronic intussusception. The apex of the invaginated bowel was formed by a small plaque no larger than the blunt end of a pencil, but the area looked suspiciously neoplastic and so an appropriate triangle of bowel and mesentery was excised. During the next two months radiotherapy failed to prevent the growth of large sarcomatous masses within the abdomen, and death occurred. This sudden end to an initial surgical success prompted a search for further facts regarding this formidable disease.

The Hospital records showed only 6 cases since 1900, and an exhaustive search of the world literature from 1852 brought the total to 106. From these reports it was found that: (1) In 90 per cent of the cases the duration of the symptoms, from the first complaint to admission to hospital, was six months or less; (2) More than 60 per cent of the patients died within six months of diagnosis.

In this paper a "clinical life-history" of these intestinal sarcomas in children is given, including an account of 2 personal cases, and the treatment is discussed.

PERSONAL CASE REPORTS

Case 1.—Gordon S., aged 3 years and 11 months, entered the Hospital for Sick Children in July, 1935, with a history of diarrhœa and vomiting seven days previously. On the day after this attack mild abdominal pain was experienced and said to be relieved by defæcation, but on admission the pain returned with increased intensity and was accompanied by many calls to stool, with foul-smelling lumpy motions and much mucus. At no time had there been rectal bleeding or any constipation.

On Examination.—The child was fairly well covered but pale; he was rolling about in colic. A large sausage-shaped lump with its concavity facing the umbilicus could be felt in the epigastrium, and as the tumour hardened with each screaming attack, it was thought to be an intussusception.

Operation.—An immediate right paramedian laparotomy displayed at the apex of the readily reduced tumour a small button-shaped disc of firm tissue, $\frac{1}{4} \times \frac{1}{4}$ in., lying rather to one side of the antimesenteric border and about eighteen inches above the ileocolic valve. The centre of the tumour was dimpled, but neither polyp in the underlying lumen nor glands in the adjacent mesentery could be felt. This disc might well have passed for an œdematous apex but for three whitish nodules, like millet seeds, embedded in its periphery. Eight inches of small bowel with a good wedge of intervening mesentery were excised, and an end-to-end anastomosis was performed.

Progress.—The child made an excellent recovery, and when Dr. Nabarro reported a "round-celled sarcoma" it was pleasant to reflect that the invagination had been the means of disclosing the early stage of such a fatal disease.

Dr. Bertram Shires was consulted, and deep-X-ray therapy was begun on the seventh day after operation. The boy was discharged on the twenty-third day and attended for further treatment.

Convalescence was marked by rapid wasting, abdominal discomfort after meals, constant diarrhœa, and much flatus. Two months after the first admission the child re-entered the ward with a twelve-hours' history of severe abdominal colic and a lump the size of an adult fist in his hypogastrium.

I was away at the time, and a laparotomy by another surgeon showed the small bowel to be invaded by many non-obstructing sarcomatous masses; "all" the mesenteric glands

were enlarged, but the liver was not involved; the abdomen was closed. The patient died at home nineteen weeks after the first symptom, and eighteen weeks after the initial resection.

Case 2.—Phyllis H., aged 5 years, was admitted to Guy's Hospital in November, 1931, suffering from abdominal pain which had occurred suddenly the previous day. During the first twenty-four hours the bowels had been opened eight times, the stools consisting only of blood and mucus. There was no vomiting.

ON EXAMINATION.—The child seemed free from pain, but a small lump the size of a walnut could be felt below the left costal margin. The lump was hard, well defined, and fixed. Pulse frequency was 102, temperature 100.5° F., and respiration rate 25. Fluroscopy showed barium flowing without obstruction round the colon to the cæcum.

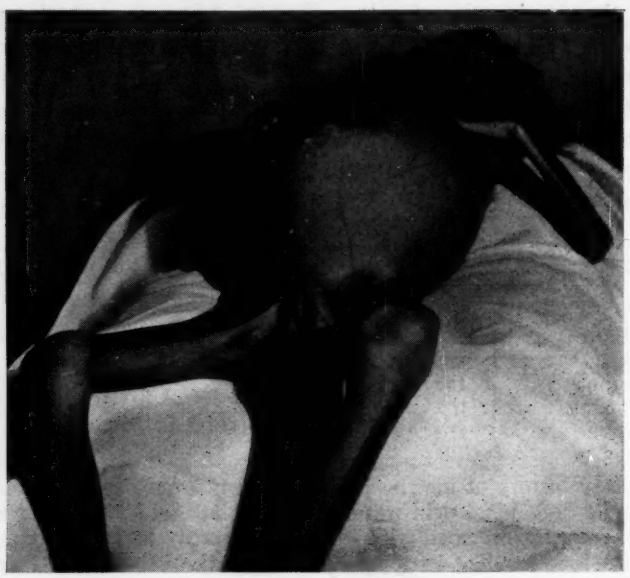


FIG. 285.—*Case* 2. Photograph showing the extensive swelling of the abdomen and the severe wasting of the arms and legs.

PROGRESS.—As the pain abated, the child was "watched" for sixteen days. She had but one slight attack of colic on the ninth day, and was finally sent to a convalescent home as a case of tuberculosis of the mesenteric lymph-glands.

RE-ADMISSION.—Six months later she was re-admitted after a bout of colic and diarrhœa. There had been mild evening pyrexias, night sweats, a slight cough, and loss of weight. The mass below the left costal margin was now the size of an orange.

OPERATION exposed a mass of hard glands in the root of the mesentery, with extensions up the posterior abdominal wall into the mesocolon and lesser sac. The original intussuscepting site could not be identified. "All" the abdominal lymph-glands were inflamed and hard, and the late Mr. R. P. Rowlands, under whose direction the laparotomy was performed, thought the glands were tuberculous. A small bunch of glands were excised and reported: "Hyperplasia of endothelium. No evidence of tuberculosis."

SUBSEQUENT PROGRESS.—A few weeks later the child went home, and nothing was heard until she returned in April, 1933. She was then severely emaciated, the abdomen was swollen (*Fig.* 285), and in the many engorged superficial epigastric veins there was a reverse flow of blood towards the thorax. A hard mass filled the left upper quadrant, and a smaller one occupied the right iliac fossa. Repeated vomiting made the retention of food exceedingly difficult, and the distended bowels responded only to enemata. Death occurred twenty months after the initial symptoms. Permission for post-mortem examination could not be obtained.

I have recently asked Professor Nicholson to look at the sections again, and in the single remaining specimen he examined there was only chronic inflammatory tissue. The history,

clinical course, and enormous abdominal distension without fistula formation strongly support the diagnosis of sarcoma. Dr. Conybeare, under whose care the child was finally admitted, was also of this opinion.

DETAILS OBTAINED FROM A STUDY OF THE LITERATURE

FREQUENCY OF CASES

As a careful search of the literature of the past eighty years yielded accounts of only 106 cases of sarcoma of the intestine in children, it is obvious that the condition is rare. The youngest age at which carcinoma of the intestinal tract has been reported in a 'white' individual is 11 years, the patient dying of cancer of the rectum. Tumours of the bowel, whether mesothelial or epithelial, are therefore no exceptions to the general rule that children are peculiarly exempt from malignant neoplasms. (*See Table I.*)

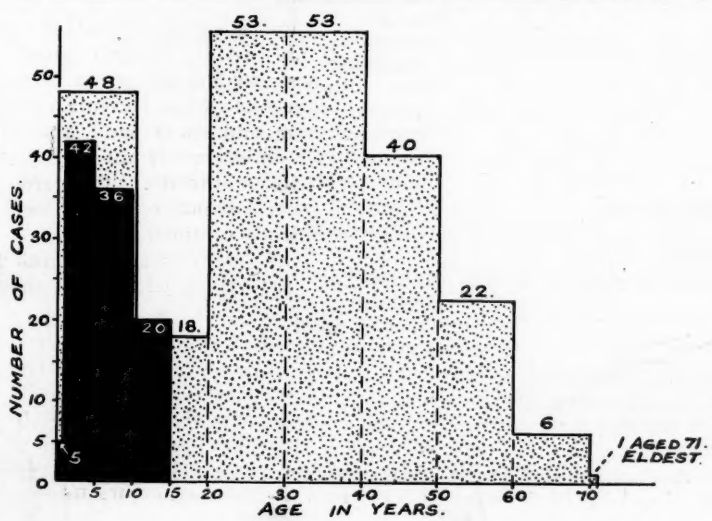
Table I.—Showing Rare Incidence of Malignant Neoplasms in Children

Author	Date	Malignant Tumours at All Ages	Malignant Tumours in Children
Libert	1851	471	15
Breslau	1883	3144	43
Nothnagel	1903	2125	0 (3 sarcomata in adults)
Warthin	1913	2000	3 (all lymphosarcomata)
Ewing	1914	52420	23 (4 under 1 year; 19 under 5 years)
	Total	60160	84

AGE INCIDENCE

Table II shows a maximum incidence of sarcoma between 1 and 5 years, infancy being singularly immune, and later childhood enjoying a transient freedom.

Table II.—Age Incidence of 103 Sarcomas in Children (black area) compared with a Separate Series of 241 Cases at all Ages (shaded area)



The 5 cases under 1 year were 2 neonates and 3 infants. Three "children" were not included as the ages were not given. Note the immunity in infancy and the steady decline from the fifth to the twentieth year.

The arrangement of the 83 children (black area) under 10 years into smaller age groups gives the 48 children (shaded area), a false numerical superiority on the chart.

When, therefore, a malignant tumour arises in a child under 11 years of age, and especially between 1 and 5 years, the balance is heavily in favour of sarcoma. Rankin has said: "Indeed, the presence of a rapidly growing tumour in the *intestine* [my italics] of a child accompanied by loss of weight, anæmia, and inanition, is strong presumptive evidence that the neoplasm is a sarcoma."

SEX INCIDENCE

Most authors state that males are affected twice as often as females, but in this series of 72 males and 26 females (sex being unstated in 8) the proportion is almost 3 to 1.

CLINICAL PICTURE

Table III shows the principal symptoms and signs in their relative frequency. Two facts are of diagnostic importance: (1) The high incidence of a palpable abdominal tumour, i.e., in 67 out of 76 stated cases. (2) The many parental complaints that the child appeared to lose ground, became "ill", "pale", "anæmic", or "very thin". Such symptoms can be conveniently grouped under the heading of "deterioration".

The 24 per cent expectation of intussusception and the comparative rarity of altered action of the bowel are also shown.

Tables IV–IX need no explanation.

Table III.—General Clinical Picture

Pain	recorded as	*Abdominal* (25 being colicky)	41	
		Right sided	12	
		Left sided	3	
		Epigastric	2	
		Lower abdominal	6	64 (31)
		No pain		11
Tumour		Palpable		67 (33)
		No tumour felt		6
		Intussusception		25
		Palpable	19	
		Impalpable, but discovered at operation	6	
		When intussusception reduced, sarcomatous apex small	9	
		Therefore tumour due to intussusception alone	9	
		Tumour due to growth alone (67 — 9)		58
Deterioration		Ill, wasted, pale, anæmic		46 (53)
		No deterioration		7
Vomiting				42 (64)
Altered Action of Bowel		Constipation	21	45 (48)
		Diarrhœa	24	
		Blood in stool	11	
		Regular	12	
Abdominal distension				47
Abdominal tenderness				19
Visible peristalsis				4
Rigidity				2

N.B.—Figures in brackets indicate the number of cases in which there is no mention of that particular symptom.

Table IV.—Duration of Symptoms

One week or under	12
One month or under	32
Two months or under	46
Three months or under	50
Six months or under	62
"Short" duration	67
Seven months or under	69
One year	4
Two years	1
Unstated	32

This table shows that in 62 out of 74 stated cases (i.e., 90 per cent) the duration of the symptoms was not longer than six months before the patient was admitted to hospital. "Short" in this case is interpreted as probably indicating a period less than six months.

Table V.—Site and Lumen

Small Intestine:		
Duodenum	3	70
Jejunum	9	
Ileum	41	
"Small"	17	
Large Intestine:		
Appendix	6	33
Cæcum	13	
Ascending colon	4	
Transverse colon	2	
Descending colon	2	
Sigmoid colon	3	
Rectum	3	
"Intestine"	3	
	106	
Lumen (where stated):		
Not obstructed by growth	34	
Obstructed by growth	8	

From this table the concentration in the ileum, cæcum, and appendicular regions will be noted.

Table VI.—Operation

Resection of Growth:		
Local removal of tumour	9	57
Small intestine to small intestine (3 deaths):		
End-to-end 6; Lateral 4; Murphy's button 1; Unstated 8	19	
Ileum to colon (no deaths)	25	
Colon to colon (3 deaths)	4	
"Laparotomy":		
Closure over inoperable growth (3 deaths)	15	18
Intussusception alone reduced	2	
Lateral anastomosis around growth	1	
No Operation (patient usually too weak)	20	
No details	11	
	106	

This table shows that in at least 38 cases (18 laparotomies and 20 where no operation was performed) the growth was too far advanced for removal.

Table VII.—MICROSCOPICAL SECTIONS AS REPORTED

Lymphosarcoma	34
"Round-cell" or "small round-cell"	41
Large round-cell sarcoma	1
Globo-cell sarcoma	1
"Lympho-blastic" sarcoma	1
"Malignant lymphoma"	1
Spindle-cell	5
"Myo-sarcoma"	1
"Giant-spindle cell"	1
"Giant-cell sarcoma"	1
"Myxo-sarcoma"	1
"Colloid sarcoma-carcinomatodes"	1
"Sarcoma"	17
	106

Table VIII.—SECONDARY DEPOSITS

No secondaries found	11	
Isolated secondary deposits in intestine	4	
Glands:		
Mesenteric	31	58
Retroperitoneal	6	
Great omentum	11	
Mediastinal	4	
Groin 3, Axilla 2, Neck 1	6	
Liver	14	
Kidney	13	
Spleen	6	
Pancreas	6	
Lung	3	
Sarcoma mass:		
Filling "whole abdomen"	4	9
Filling pelvis	5	
Odd secondaries:		
Skin of abdomen	5	12
Skin of thigh	1	
Nipple	2	
Sternum	1	
Rib	1	
Hernia sac	1	
Epididymis	1	
Perforation	4	

The high incidence of glandular deposits is contrary to the general rule that sarcomata, except those arising in the tonsil or testicle, and melanomas, do not metastasize to lymph-glands.

Table IX.—END-RESULTS OF 106 CASES

1. DEATHS:			
Stated		68	74
Presumed from:			
prognosis unfavourable	2	6	
local removal only	2		
partial removal	1		
perforation of growth	1		

Table continued on p. 435

2. SURVIVALS :		
Of short duration—		
2 cases of 1 month; 1 each of 2, 3, 5, 6, 9, 10 months	8	
Of one year or more—		
1 case of 1 year; 2 cases of 18 months; 2 cases of 2 years; 1 case of 3 years; 1 case of 4 years; 2 cases of 7 years	9	17
3. UNSTATED		15
		106

This table shows that *at least* 74 out of 91 stated cases (i.e., 81 per cent) were fatal. As 8 further patients were not followed up for a year it is probable that some of these also died. Only 9 patients (8 per cent) lived one year after the disease had been diagnosed, and from this angle the mortality rises to 92 per cent. It is therefore safe to say that the disease is exceeding fatal.

DISCUSSION

DIAGNOSIS

Early diagnosis depends upon a clinical picture that can be recognized at the commencement of the disease, yet three difficulties immediately present themselves :—

1. Children of a very young age are affected, and cannot be expected to draw attention to early symptoms.

2. A small lump developing in a large body cavity is sheltered from early diagnosis.

3. The growing sarcoma usually extends round the bowel wall, expanding rather than contracting the lumen, so that even intestinal obstruction rarely discloses the nature of the lesion. When it does so the growth is usually so large that the value of the obstruction, as a diagnostic sign, is lost.

Even the 24 per cent probability of an acute intussusception unmasking the disease does not lessen these difficulties, for in 10 cases (19 — 9, *Table III*), i.e. 9·5 per cent, the growth itself was already palpable, and therefore of a dangerous size, before the intussusception occurred: in only 9 cases (8·5 per cent) could it be claimed that the telescoping drew attention to the disease at an early stage. In 8 cases, 7·5 per cent (*Table V*), the growth actually obstructed the bowel lumen, but this, as explained above, cannot be utilized as an early sign. Visible blood in the stool, 10 per cent (*Table III*), and tenderness or rigidity must be regarded as late signs. *Table III* also emphasizes the importance of searching for an abdominal tumour in a child who suddenly " deteriorates " without obvious cause, especially if the stomach-ache has been sporadically experienced. An anæsthetic with gentle bimanual examination helps to detect such neoplasms.

Accessory diagnostic methods include a barium meal and enema, the latter to exclude a colonic lesion. In expert hands the methods of Abbot and Johnston may be employed: in this a small double-lumen tube is swallowed, the balloon on one of the adjacent ends is inflated after the pylorus has been passed, and peristalsis is allowed to carry the bag down to the level of the tumour, where it is arrested. A thin solution of barium is then run down the other lumen for immediate X-ray exposure of any deformation.

Examination of the stool for occult blood is important, for Joyce affirms a positive reaction in 100 per cent of cases of malignant neoplasm of the intestine. He does not, however, state the number of cases on which this statement is based.

TREATMENT

The prevalence of glandular metastases and the rapid demise of my first case, where eight inches of bowel and accompanying triangle of mesentery had been excised, suggest that the resection should be wider and that the glands in the root of the mesentery should be removed in the same careful way that the axilla is dissected in a radical Halstead operation for carcinoma of the breast, or when a melanotic 'sarcoma' has been found in the arm.

A wide one-stage resection followed by a blood transfusion is well tolerated in children, and is obviously without the danger of dissemination that attends a two-stage procedure. Any anastomosis to the colon is best accompanied by a thin rectal tube passed up the bowel through the stoma, and I have employed this with advantage in children for anastomoses as high as the hepatic flexure.

Deep X-ray therapy has been given to a few cases, but it is difficult to assess its value, only a few out of 17 survivals having been treated in this way. The rapid decline of my first patient, who received adequate dosage, cannot be attributed, for reasons already given, to a failure of this therapy.

SUMMARY

1. An analysis is given of 106 cases of sarcoma of the small and large intestine in children.

2. Reports are given of personal cases.

3. A clinical picture is given which emphasizes that illness, pallor, or wasting occurring in a child without obvious cause, should suggest a sarcoma, and that the abdomen should be carefully searched for a tumour.

4. A palpable tumour is to be expected in at least 63 per cent of the cases.

5. The disease runs a rapid course, the symptoms seldom lasting more than six months before operation is undertaken.

6. More than 60 per cent of children die within six months, and 92 per cent within twelve months, of diagnosis.

7. Treatment consists in early diagnosis and wide resection, with deep dissection of the mesenteric glands including the root of the mesentery.

8. The prognosis is always grave.

My thanks are due to my surgical colleagues at the Hospital for Sick Children for allowing me to search their records; to Guy's Hospital for permission to publish *Case* 2; to Professor Nicholson for advice in pathology; and to the librarians of the Royal Society of Medicine for their excellent translations and their unfailing kindliness.

BIBLIOGRAPHY

ABALLI, A. A., *Bol. Soc. Cub. de Pediat.*, 1929, i, 497.
ABBOT and JOHNSTON, *Surg., Gynecol. and Obst.*, 1938, lxvi, 691.
ABBE, R., *Ann. of Surg.*, 1895, xxi, 592.
APITZ, K., *Zentralb. f. allg. Pathol. u. pathol. Anat.*, 1933, lvii, Sonderbd. 1.

ARNOTT, H., *Trans. Pathol. Soc. Lond.*, 1874, xxv, 150.
BALTZER, M., *Arch. f. klin. Chir.*, 1892, xliv, 731.
BARACZ, R. VON, *Ibid.*, 1891, xlii, 501.
BARLING, G., *Ann. of Surg.*, 1907, xlv, 242.
BATTLE, W. H., *Lancet*, 1920, i, 905.
BESSEL-HAGEN, F., *Arch. f. pathol. Anat.*, 1885, xcix, 99.
BOKAY, Z. VON, *Ibid.*, 1913, ccxiii, 249.
BOUDET and RIMBAUD, *Arch. Soc. de Sci. méd. et biol. de Montpellier*, 1928, ix, 68.
BRESLAU, *Arch. f. pathol. Anat. u. Physiol.*, 1863, xxviii, 556.
BRINKMAN, *St. Agnes Hospital, Philadelphia*, 1929, Nov. 16 (quoted by FRIEND).
BRUN, V., *Riv. di Clin. Ped.*, 1912, x, 465.
CHARACHE, H., *Amer. Jour. Surg.*, 1935, xxvii, 171.
CAPECCHI, E., *Policlinico* (Sez. chir.), 1927, xxxiv, 153.
CHARLEUX and CUNY, *Bull. Soc. de Pediat. de Paris*, 1934, xxxii, 428.
CIOCCA, quoted by SIMON.
CLOPTON, M. B., *Jour. Missouri Med. Assoc.*, 1920, xvii, 361.
COMBA, C., *Clin. mod.*, 1901, vii, 20.
CONCETTI, *Sez. tosc. Soc. ital. Pediat.*, 1903 (quoted by FOA).
CORNER and FAIRBANK, *Trans. Pathol. Soc. Lond.*, 1905, lvi, 20.
CONDAT, *Arch. de Méd. des Enf.*, 1921, xxiv, 679.
CRAWFORD, F. B., *Med. Jour. of Australia*, 1926, i, 553.
CUIZZA, R. S., *Pediat. d. med. prat.*, 1934, ix, 658.
DEBRUNNER, *Inaug. Diss.*, Zurich, 1882.
DIXON, C. F., *Western Jour. Surg.*, 1934, xlii, 216.
DULIZKI, *Pedia.* 1912 (quoted by MIROTWORZEW and SACHAROW, *Arch. f. klin. Chir.*, 1924, cxxx, 256).
EGTERMEYER, *Inaug. Diss.*, Greifswald, 1920 (quoted by BAGOZZI, U.C., *Tumori*, 1934, xx, 110).
EISELSBERG, F., *Zeits. f. Kinderheilk*, 1935–36, lvii, 361.
EWING, J., *Neoplastic Diseases*, 1928.
FANUCCI, M., *Policlinico* (Sez. chir.), 1930, xxxvii, 53.
FISHER, T., *Bristol Med.-Chir. Jour.*, 1901, xix, 29.
FLEXNER, S., *Johns Hopkins Hosp. Rep.*, 1893, iii, 153.
FOA, A., *Pediat. d. med. prat.*, 1933, viii, 704.
FRIEDJUNG, E., *Gesellsch. f. inn. Med. u. Pediat.*, 1903 (quoted by FOA).
FRIEND, E., *Illinois Med. Jour.*, 1926, July, 50.
FULLERTON, A., *Brit. Jour. Surg.*, 1925–26, xiii, 754.
GAZA, quoted by SIMON.
GOODMAN, A. L., *Arch. of Pediat.*, 1916, xxxiii, 721.
GRAHAM, C. R., *Brit. Med. Jour.*, 1916, ii, 801.
GRANCLAUDE, LAMBRET, and DRIESSENS, *Ann. d'Anat. pathol.*, 1933, x, 220.
GRAVES, S., *Jour. Med. Research*, 1919–20, xl, 415.
GRAY, J., *Jour. Pathol. and Bacteriol.*, 1934, xxxviii, 7.
HAGGARD, quoted by SIMON.
HARTE, R. M., *Ann. of Surg.*, 1900, xxxii, 616.
HEBB, SYERS, and HERBERT, *Westminster Hosp. Rep.*, 1888, iii, 187.
HELMHOLZ, H. F., *Proc. Internat. Assemb. Inter-State P.-G.M.A. N. Amer.*, 1931, 209.
HOMANS, J., *Ann. of Surg.*, 1896, xxiv, 42.
HORN, quoted by ORTH, L., *Ueber die Sarkome des Darms*, 1890. Tübingen.
JALLAND, *Lancet*, 1894, i, 1007.
JOHANNESSEN, A., *Acta pædiat.*, 1928, vii, supp. II, 213.
JOPSON and WHITE, *Amer. Jour. Med. Sci.*, 1901, cxxii, 815.
JOYCE, *Ann. of Surg.*, 1934, c, 949.
KEETLEY, C. B., *Lancet*, 1896, ii, 230.
KOCH, E., *Deut. Zeits. f. Chir.*, 1925, cxci, 376.
LANELONGUE, *Jour. de Méd. de Bordeaux*, 1897, xxvii, 495.
LECÈNE, P., *Thèse de Paris*, 1904 (quoted by MALBRAND, *Thèse de Paris*, 1922).
LEVEUF and GODARD, *Ann. d'Anat. pathol.*, 1936, xiii, 1067.
LEBERT, H., *Traité pratique des Maladies cancéreuses*, 1851, 141. Paris.
LIBMAN, E., *Amer. Jour. Med. Sci.*, 1900, cxx, 309.
LINN, F. W., *Trans. Central States Pediat. Soc.*, 1927, ii, 49.
LIU, J. H., *Arch. of Surg.*, 1925, xi, 602.
LORIA, F. L., *New Orleans Med. and Surg. Jour.*, 1925–26, lxxviii, 201 (quoted by FOA.)
MACERA, *Arch. latino-amer. de Pediat.*, 1911 (quoted by FOA).
MACKID, L. S., *Canad. Med. Assoc. Jour.*, 1916, vi, 325.
MADELUNG, *Zentralb. f. Chir.*, 1892, xix, 617.

MAGNUSSON, R., *Acta chir. Scand.*, 1933–34, lxxiii, 576.
MOLTENI, P., *Clin. pediat.*, 1932, xiv, 197 (quoted by FOA).
NOTHNAGEL, H., *Der specielle Pathologie und Therapie*, 1903, xvii, 340.
PALUMBO, E., *Riforma Med.*, 1933, xlix, 1727.
PATTISON, A. C., *Arch. of Surg.*, 1934, xxix, 907.
PEPIN, C. J., *Jour. de Méd. de Bordeaux*, 1890–91, xx, 520.
PERRY, C. L., *Proc. Staff. Meet. Mayo Clin.*, 1929, iv, 197.
PICOT, C., *Rev. méd. de la Suisse rom.*, 1883, iii, 660.
POWER, D'ARCY, *Clinical Jour.*, 1912, xl, 191.
RANKIN, F. W., *Colon, Rectum, and Anus*, 1932, 446.
RENNER, quoted by SIMON.
ROBERTSON and GIVEN, *Proc. Pathol. Soc. Phil.*, 1900–01, N.S. iv, 268.
ROHDENBURG, G. L., *Proc. N.Y. Pathol. Soc.*, 1919, xix, 2.
ROVSING, T., *Hospitalstid.*, 1897, ix, 1085.
SACCONE, A., *Arch. latino-amer. de Pediat.*, 1912, v, 375 (quoted by FOA).
SALA SÁNCHEZ, *Anal. de Pediat.*, 1935, ii, 627.
SANTAS, quoted by SIMON.
SARTORELLI, E., *Policlinico* (Sez. prat.), 1928, xxxv, 1504.
SIEGEL, E., *Berlin. klin. Woch.*, 1899, xxxvi, 767.
SIMON, H., *Die Sarkome*, 1928. Bd. 43 of *Neue Deutsche Chirurgie.*
STERN, C., *Berlin. klin. Woch.*, 1894, xxxi, 802.
TELLING, W. M., *Brit. Jour. Child. Dis.*, 1920, xvii, 192.
THIERFELDER, U., *Inaug. Diss.*, Leipzig, 1909, 6.
TSCHERNOW, *Detskaja Med.*, 1905 (? 1906) (quoted by MIROTWORZEW and SACHAROW, *Arch. f. klin. Chir.*, 1924, cxxx, 256).
ULLMAN, A., *Ann. of Surg*, 1932, xcv, 878.
VAN ZWALENBURG, C., *Jour. Amer. Med. Assoc.*, 1901, xxxvi, 617.
WARREN, J. C., *Boston Med. and Surg. Jour.*, 1898, cxxxviii, 177.
WARTHIN, A. S., *Arch. of Internal Med.*, 1915, xv, 444.
WHARTON, H. R., *Internat. Clinics*, 1894, 4th series, ii, 199.
WHEEDON, W. M., *Ann. of Surg.*, 1929, xc, 247.
WINTERNITZ, F., *Wien. klin. Woch.*, 1922, xxxv, 293.
WOLKOWITSCH, *Verhandl. d. chir. Gesellsch. zu Kiew*, 1915 (quoted by MIROTWORZEW and SACHAROW, *Arch. f. klin. Chir.*, 1924, cxxx, 256).
WORTMAN, W., *Deut. Zeits. f. Chir.*, 1913, cxxiii, 103.
WRIGHT, R. B., *Amer. Jour. Cancer*, 1935, xxiv, 65.
YOVTCHICH and STAINOVITCH, *Bull. et Mém. Soc. nat. de Chir.*, 1933, lix, 1131.

SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF SARCOMA IN THE INFRASPINATUS MUSCLE

By CECIL P. G. WAKELEY, London

Sarcoma originating in the muscular system must be considered to be among the rarer forms of this disease. The following case is of interest because the clinical diagnosis appeared to be that of periosteal sarcoma of the scapula.

History.—John M., aged 17, a naval rating, was admitted to King's College Hospital on May 11, 1937, complaining of a lump on the right shoulder, which was noticed and pointed out to him by his mates three weeks previously.

On Examination.—The patient was a well-developed and muscular individual. A smooth, firm, well-defined swelling could be seen and felt in the right infraspinous fossa. It was rounded in appearance and approximated to the size of an orange (*Fig.* 286). It was attached to the scapula and moved with all movements of that bone. The swelling was quite painless and the skin over it could be moved in all directions. There was no limitation of movement of the arm whatever. The patient felt quite fit and was otherwise perfectly well. The heart and lungs were normal and there was no evidence of any mediastinal deposits. Radiographs of the chest did not reveal any abnormality, nor was there any evidence of periosteal involvement of the scapula.

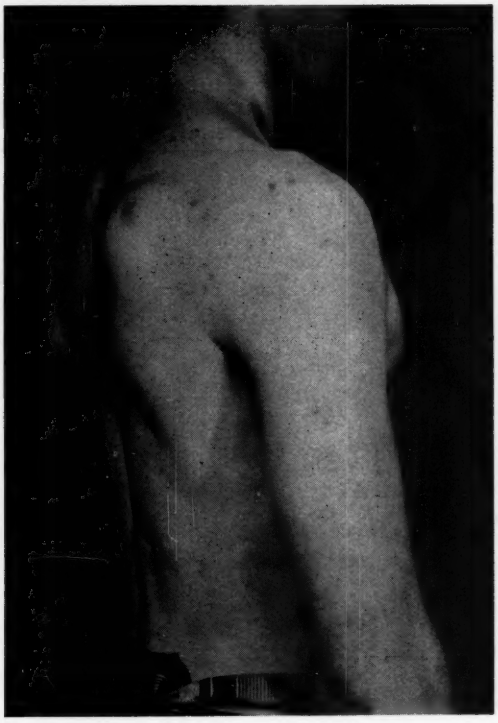


Fig. 286.—Photograph of patient, showing tumour in the region of the right scapula.

Operation.—Excision of the tumour, together with the scapula, was advised and carried out under gas and oxygen anæsthesia on May 19, with the patient lying prone on his face. An H-shaped incision was made over the scapula, and the flaps of skin thus outlined were reflected back. All the muscles which were attached to the margins of the scapula were then detached, and the posterior scapular and subscapular arteries secured and ligatured. The bone with the

supraspinatus and infraspinatus muscles was removed by cutting through the capsule of the shoulder-joint. The humeral attachment of the shoulder-joint was then united by means of silk sutures to the under surface of the clavicle. The muscles were united so that the trapezius was continuous with the deltoid. The skin was closed with interrupted silkworm gut sutures, a small rubber drain being inserted into the lower part of the wound.

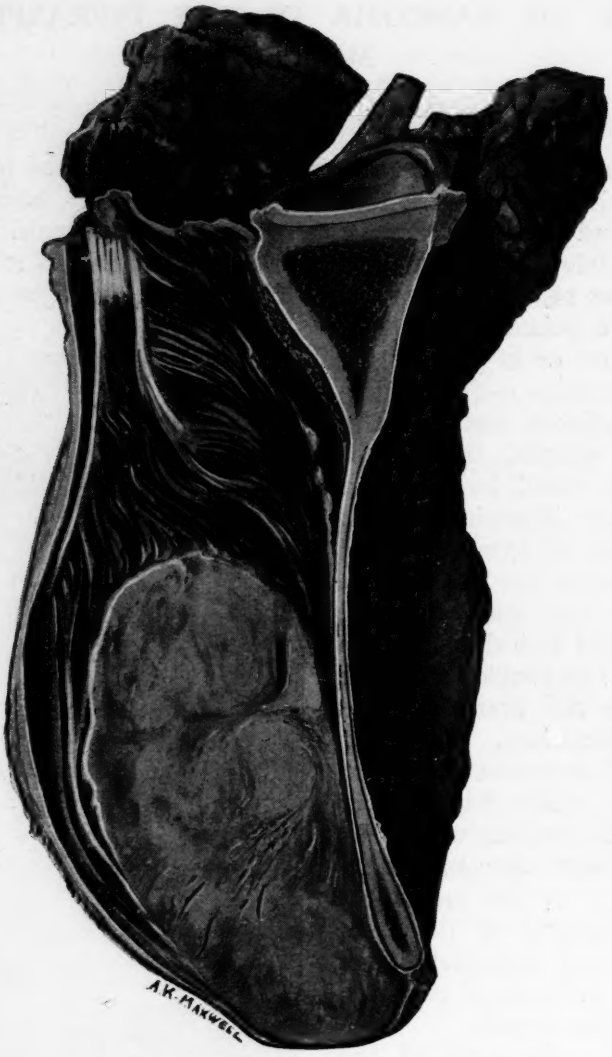


Fig. 287.—Section of tumour and scapula.

The patient made a rapid recovery from the operation, and was discharged after sixteen days. Massage and electrical treatment to the muscles around the shoulder were instituted three weeks after the operation.

PATHOLOGY.—A section was made through the excised scapula and a white circumscribed encapsuled tumour, $2\frac{1}{2}$ by $1\frac{1}{2}$ in., could be seen (*Fig.* 287) occupying the infraspinatus muscle. The tumour is firmly attached to the bone and to the

muscle sheath in the neighbourhood of the vertebral border, but the periosteum is not involved, nor is the underlying bone affected. The tumour appears to have arisen from the fascial sheath and to have displaced the muscle fibres during its growth. Histologically the growth is a spindle-celled sarcoma (*Fig.* 288). Sections show a highly vascular cellular growth with numerous mitotic figures. There is no special arrangement of the cells other than a tendency to concentrate around the blood-vessels. The cells have large round or oval reticular nuclei with scanty cytoplasm.

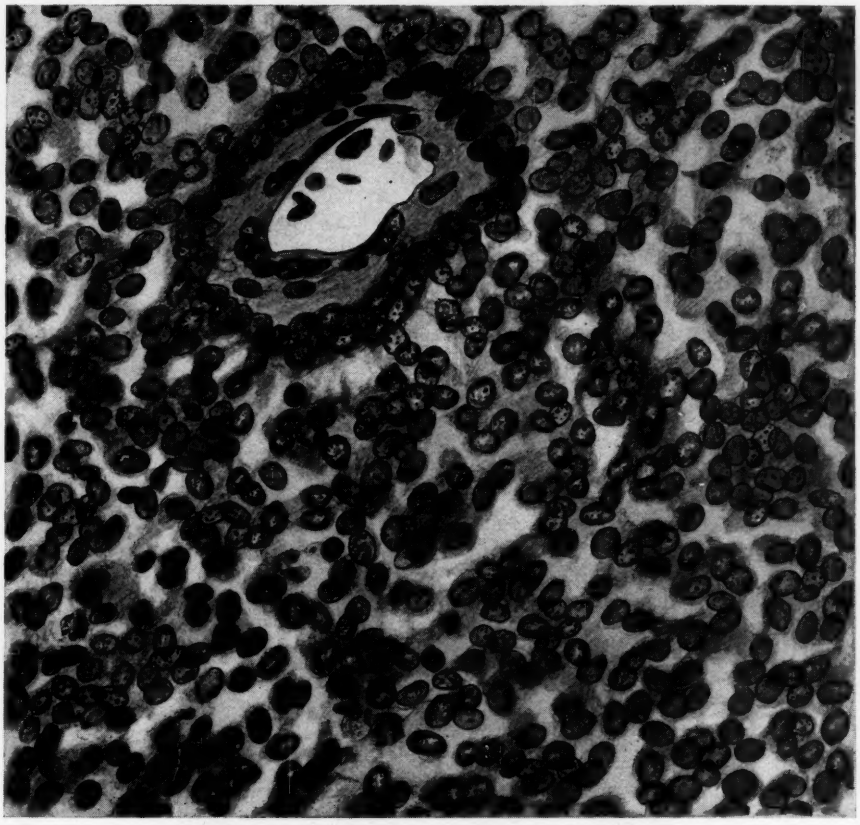


Fig. 288.—Microscopical section of tumour showing the type of cell present. (× 600.)

SUBSEQUENT PROGRESS.—The patient was examined again in October, 1937, and was found to have practically no disability. The only limitation of movement was abduction of the arm. He could ride a bicycle, play tennis, and use a typewriter. X-ray examination of the chest showed no abnormality apart from the absence of the scapula and the situation of the right humerus under the outer end of the clavicle (*Fig.* 289).

The patient was again examined in May, 1938, just over a year from the date of excision of the scapula. He was doing full work as a clerk and using a typewriter for more than four hours a day. He played tennis twice a week, using the racquet in his right hand. The movements of the shoulder were good and abduction to a right angle could be obtained with an effort (*Fig.* 290). The patient was doing

exercises each day and had commenced swimming before breakfast. He had no difficulty in doing the 'crawl'. There were no signs of any secondary deposits in the lungs, and the patient was fit in every way, with a normal blood-count.

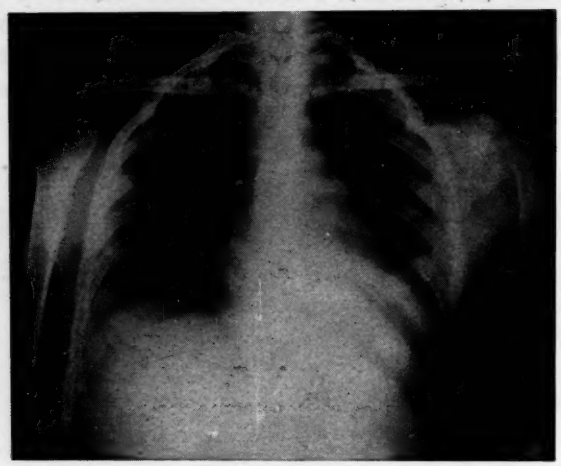


FIG. 289.—Radiograph of patient showing the relation of the right humerus to the clavicle.

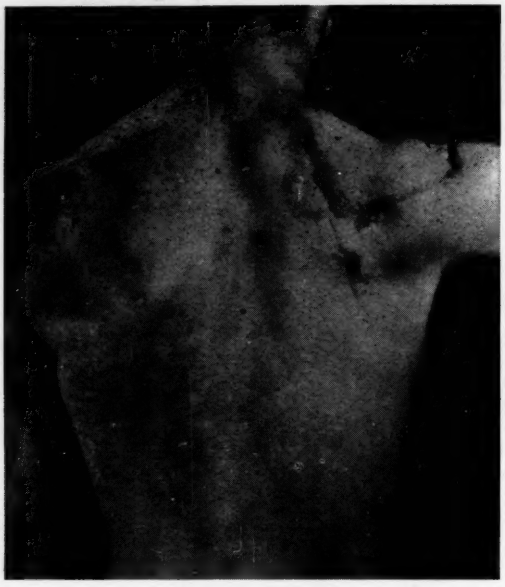


FIG. 290.—Photograph of patient one year after operation, showing the amount of abduction at the shoulder-joint.

Few cases of excision of the scapula are recorded, and it is not sufficiently recognized how little disability such an operation produces, especially if the patient co-operates well in the after-treatment.

The specimen is now in the Museum of the Royal College of Surgeons, and I wish to thank the Pathological Staff of the College for the help given me in examining this interesting tumour.

A CASE OF BENIGN TERATOMA OF THE TESTICLE IN AN INFANT

By CECIL P. G. WAKELEY, London

Testicular tumours are rare in infancy, and it is because of this that the following case is recorded.

History.—Mervyn F., aged 3½ years, came under observation in January, 1937, being referred to me as having a vaginal hydrocele on the left side.

On Examination.—There was a definite swelling in the left side of the scrotum about the size of a large walnut. It was definitely oval, cystic, and translucent. There was no difficulty in getting above the swelling, but it was not reducible. The left testicle could be felt below the swelling, and the case therefore appeared to be a straightforward one of hydrocele of the tunica vaginalis.

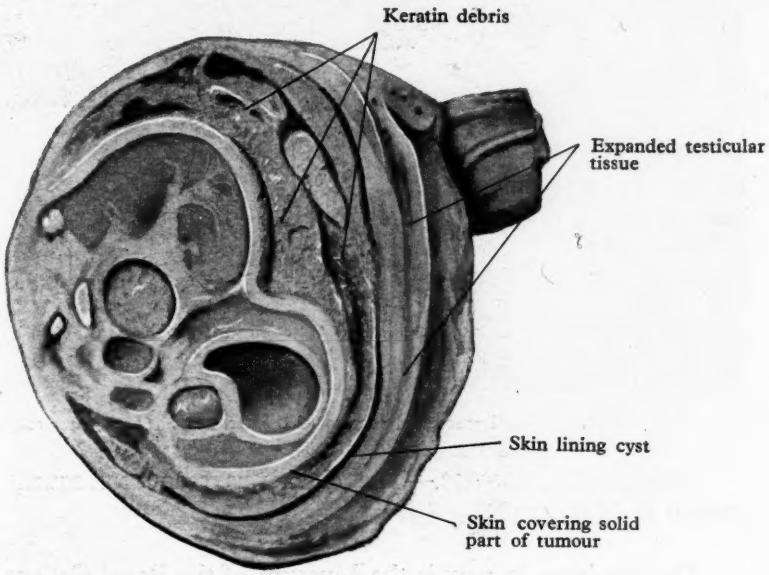


Fig. 291.—Section of teratoma of testicle. (× 1½.)

The child was kept under observation, as it was considered that in all probability the hydrocele would gradually disappear. In May, 1937, the hydrocele was definitely smaller, but the testicle itself was larger than the other side. In July, 1937, no hydrocele could be detected, but the testicle was three times the size of the right one. It was therefore decided to remove the testicle as it was considered to be a solid tumour involving the organ.

Operation.—This was undertaken on July 15, through an inguinal incision, and an uneventful recovery followed.

Pathology.—On section, the specimen consisted of a tense spherical tumour, 1½ by 1¼ in., with 1 in. of vas attached to one pole (*Fig.* 291). A cyst containing sebaceous material could be seen, and this was almost completely filled by a rounded nodule covered with skin on its free surface. The nodule contained numerous small cysts, yellow fatty areas, and cartilage.

Histologically, the sections showed a cyst wall composed of a thin layer of compressed testicular tissue, and lined in part by squamous epithelium. The enclosed nodule was covered with skin containing hair follicles and sebaceous glands, and was composed of vascular connective tissue and plain muscle, with several nodules of cartilage. There were numerous small cysts and tubules lined by squamous, columnar, or transitional epithelium, and mucous glands of the salivary type (*Fig.* 292), the whole appearance being consistent with that of a benign teratoma.

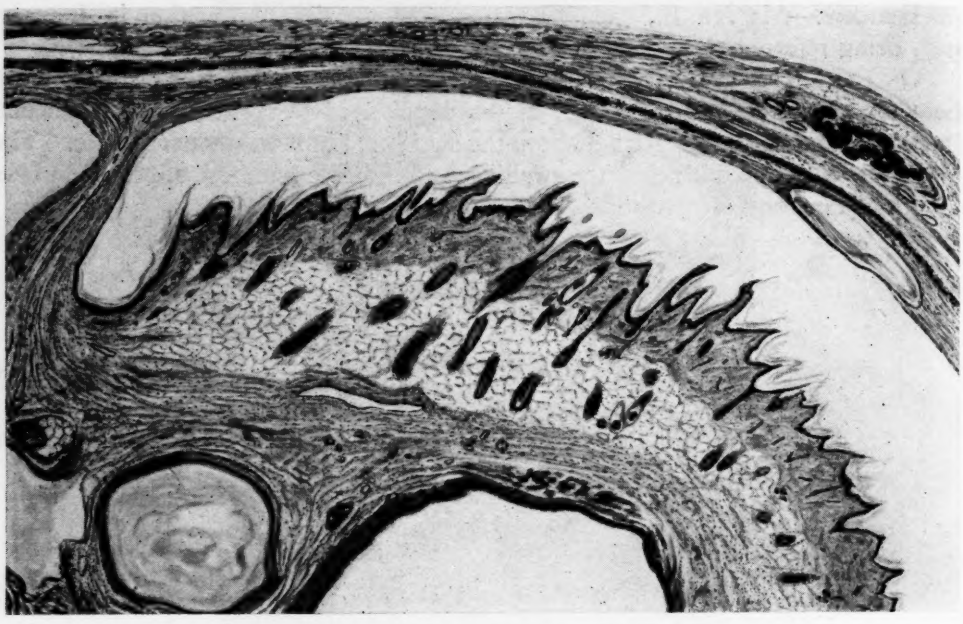


FIG. 292.—Microscopical section of the teratoma. (*Low power magnification.*)

SUBSEQUENT PROGRESS.—The child was seen at frequent intervals and was quite fit in May, 1938.

The specimen in now is the Museum of the Royal College of Surgeons, and I wish to thank the Pathological Staff of the College for their help in the histological investigation of this case.

A CASE OF INTUSSUSCEPTION OF THE NORMAL APPENDIX INTO THE CÆCUM

BY G. T. MOWAT, GLASGOW

CASES of this type of intussusception are sufficiently rare to be worthy of notice.

HISTORY.—F. M., a robust young slater, aged 19, was admitted to the Glasgow Royal Infirmary on October 9, 1937. One week prior to admission he was seized with fairly acute pain referred to the centre of the abdomen, colicky in type, but which after two days settled into the right iliac fossa and remained there until his admission. There was no history of previous abdominal discomfort or of other illness.

On Admission.—The temperature and pulse were normal. The abdominal wall generally was lax and free from distension. Slight tenderness and very slight rigidity were present on pressure in the right iliac fossa, and the clinical picture was one of a subacute appendicular attack which was not progressing. There was no vomiting and he was quite comfortable in bed.

On October 11 a gridiron incision was made, and the area of the cæcum to which the appendix is normally attached was found to be replaced by a dimple into which the appendicular mesentery ran. The appendix could be felt inside the cavity of the cæcum. The cæcum was incised, revealing a completely inverted appendix, 4 in.

Fig. 293.—Diagrammatic representation of the intussusception. The new lumen, shown empty, was filled with the appendicular mesentery.

long, the mucous membrane being granular and congested, with areas of commencing gangrene in its terminal inch (*Figs.* 293, 294). It was ligated and excised, and the opening in the cæcum was closed. Recovery was uneventful.

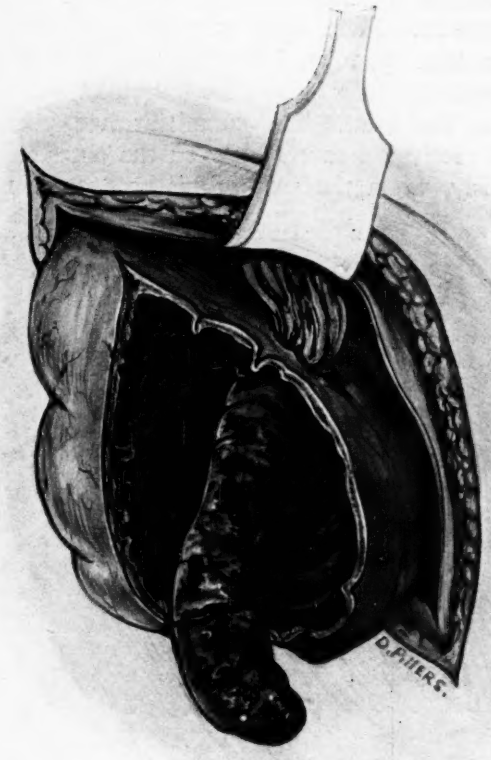


Fig. 294.—A view of the appendix from inside the cæcum, showing the extreme congestion and small gangrenous patches near the tip.

Histological Examination.—Acute inflammation and congestion only. There was no other abnormality.

Comment.—The following points are of interest :—

1. The liability to intussusception of the thin-walled, wide-channelled, atavistic appendix, occasionally seen in adults, and which must have been present in this case, the 'turn-coat' peritoneal lumen holding the mesentery with ease.

2. The close resemblance of its symptoms and clinical features to those of a subacute attack of appendicitis.

I am indebted to Dr. Tina Gray and Miss Pillars for the accompanying illustrations.

A CASE OF CALCIFICATION IN THE EPICONDYLAR BURSA AT THE ELBOW

By A. R. HAMILTON, Sydney

In view of the well-established clinical entity 'calcification in the subdeltoid bursa', the occurrence of a similar condition in a bursa about the external epicondyle of the humerus is probably worth recording.

History.—Male, aged 42 years; plumber.

On Aug. 23, 1937, I saw this man on account of a complaint of pain and stiffness about the outer side of his left elbow and in the extensor muscles immediately distal to that joint. The complaint had been present a week. He stated that he had received a severe blow on the outer side of his left elbow eleven months previously, when the spar of his boat struck him during a collision with another

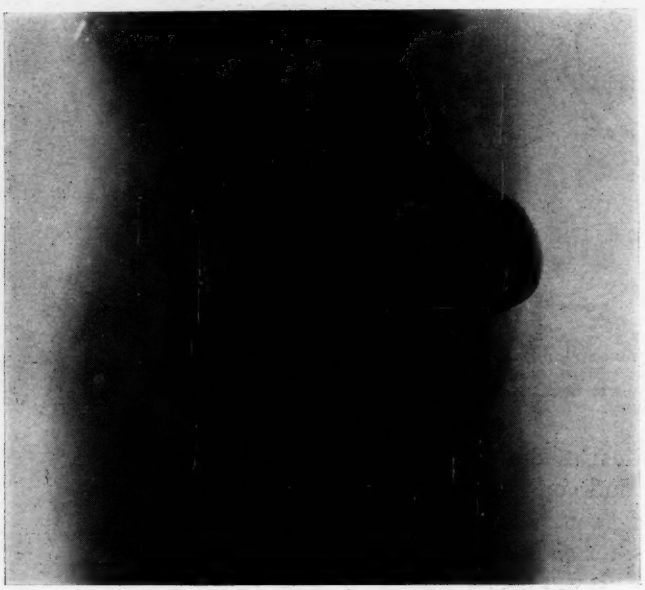


Fig. 295.—Radiograph showing calcium deposit on the radial side of the elbow-joint.

vessel. The elbow was "sore" for a few days at that time, but "settled down" and did not trouble him until a week before I saw him.

On Examination (Aug. 23, 1937).—There was considerable swelling about the external epicondyle of the left humerus and great increase of local temperature (to palpation). The epicondyle was exquisitely tender to touch. Movements of the elbow were very limited—flexion to a right angle and extension to 155°.

A number of infected teeth were present.

X-ray examination at that time revealed a deposit of calcium on the radial side of the elbow-joint (*Fig.* 295).

Treatment.—(1) The hand, forearm, and lower half of the upper arm were encased in plaster-of-Paris, the hand in dorsiflexion and the elbow at a right angle.

This was worn for four weeks, when (2) A short 'cock-up' splint was applied to the wrist, and a 'collar and cuff' was worn for about two weeks.

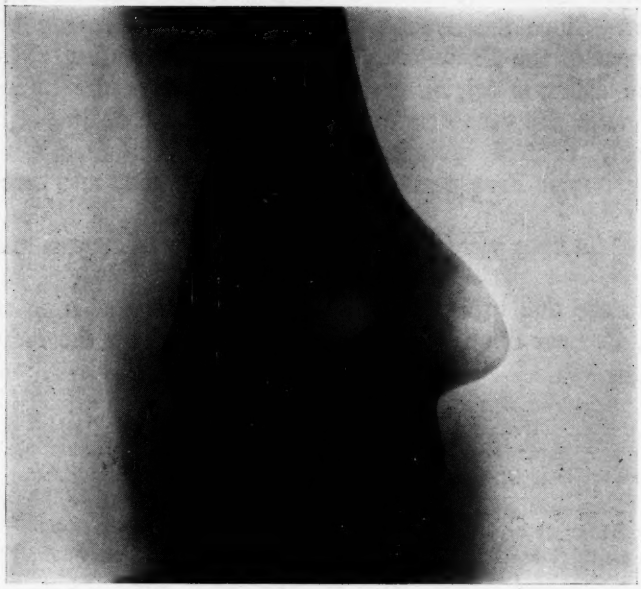


Fig. 296.—Radiograph showing the condition some five weeks later. Nearly all the calcium deposit has been absorbed.

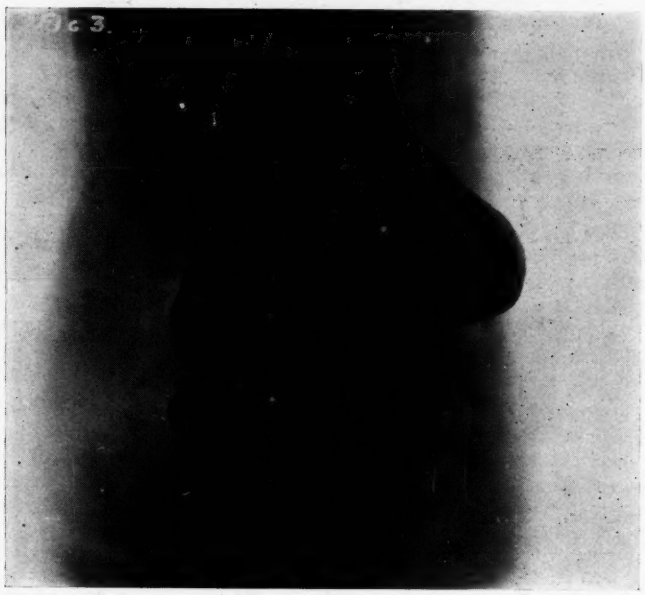


Fig. 297.—The condition seven months after the commencement of treatment. The calcium deposit has entirely disappeared.

PROGRESS.—On Sept. 29, 1937, tenderness was very slight and there was no swelling. X-ray examination showed that practically all of the calcium had been absorbed (*Fig.* 296).

On March 18, 1938, there was no pain, tenderness, or swelling. The patient had had no pain during the preceding four months and was doing his usual work. X-ray examination showed complete absorption of the calcium deposit (*Fig.* 297).

Comment.—Since the deposit was too laterally placed to be in the radial collateral ligament of the elbow-joint, it must have been either (1) in the substance of the common extensor origin, or (2) in a bursa over the external epicondyle. If the former were the case one would expect a more diffuse distribution rather than the sharply defined globular appearance of the deposit as seen in the radiograph (*Fig.* 295). For that reason it must be regarded as a case of calcification in a bursa.

The clinical findings were those of a tennis elbow of an extremely severe type.

My thanks are due to Dr. F. G. N. Stephens for referring the case to me, and to Dr. H. R. Sear for his interest in the case and for the radiographs.

REVIEWS AND NOTICES OF BOOKS

A Text-book of X-ray Diagnosis. By British Authors. Edited by S. COCHRANE SHANKS, M.D., Physician with Charge of Out-patients, Departments of Radiology and Electro-therapy, Charing Cross Hospital; Radiologist, the Prince of Wales's General Hospital, etc.; PETER KERLEY, M.D., M.R.C.P., D.M.R.E., Physician to the X-ray Department, Westminster Hospital; Radiologist, Royal Chest Hospital, etc.; and E. W. TWINING, M.R.C.S., L.R.C.P., D.M.R.E., Radiologist, Royal Infirmary, Manchester; Radiologist, Christie Hospital; late Hunterian Professor, Royal College of Surgeons, etc. In three volumes. Cr. 4to. Vol. I, pp. 598 + xii, with 398 illustrations. Vol. II, pp. 546 + xii, with 307 illustrations. 1938. London: H. K. Lewis & Co. Ltd. Vol. I, 50s.; Vol. II, 42s. net.

IN recent years X-ray diagnosis has invaded the fields of more and more branches of medicine and surgery, with a result that specialization has arisen within diagnostic radiology itself. The editors of this new work have therefore obtained the collaboration of a group of radiologists and clinicians to deal specifically with those branches of the subject in which they are especially proficient, and the result is a work which will probably remain the most authoritative text-book on diagnostic radiology in English for many years to come.

Volume I deals mainly with the thorax, including sections on the cardiovascular system and the respiratory system, and ends with a section on the genito-urinary tract. These subjects are very fully dealt with, with the mention of only such technical details as are essential to the adequate examination and proper interpretation of the radiological findings. The impression gained from a study of the first volume is that the authors are primarily clinicians, physicians, and surgeons, and secondarily radiologists.

In Part I Dr. Kerley deals capably with the cardiovascular system, nowadays a very extensive subject, and includes and evaluates the more special methods of examination, such as kymography, X-ray cinematography, etc. He has selected an excellent group of illustrations, and the subject is most competently handled, with many references to the work of leading cardiologists, both British and foreign.

Part II, on the respiratory system, by Dr. Twining occupies some 367 pages. It is very well done and reveals the work of a painstaking clinician of wide knowledge and experience and shrewd judgment. This section is also illustrated with superb radiographs and diagrams most carefully selected, and a very extensive bibliography is appended.

Mr. Jennings Marshall and Dr. Cochrane Shanks are responsible in Part III for the section on the urinary and male genital tracts. This collaboration of surgeon and radiologist has resulted in a well-balanced and lucid description of this most important branch of medicine and surgery.

Volume II of this work maintains the high standard attained in *Volume I*. It deals mainly with the X-ray examination of, and diagnostic problems connected with, the abdomen.

The alimentary tract is dealt with very completely in four sections by Dr. Cochrane Shanks, who has drawn on his own wide experience in this work and has also obtained helpful contributions from co-workers both at home and abroad. Recent methods of examination of the mucosal relief of the whole tract by the use of thorotrast and similar colloidal preparations, both with and without the additional use of air to provide a double contrast, are included. The illustrations are first-class, and the clinical aspects of the subjects are never lost sight of.

In Part II, Dr. Kerley contributes a most interesting article on the biliary tracts, including some quite recent work of his own and others on the functional disorders of the biliary system and the visualization of the bile-ducts. Some important details of technique in the preparation and examination of the patient under investigation are necessarily included, but the technical aspects are always kept subservient to the main clinical problems.

In Part III Dr. Cochrane Shanks adds a short chapter on the liver, spleen, pancreas, and adrenals.

In Part IV, which is entitled " The Female Genital Tract ", Dr. R. E. Roberts and Mr. J. St. George Wilson convincingly demonstrate the value of X rays in the investigation of the female pelvic organs, using injections of gas and an opaque medium.

The final section by Dr. R. E. Roberts is devoted to the subject of obstetrics. Dr. Roberts has had considerable experience in this branch of radiodiagnosis, and his original work on pelvimetry is already well known. The work of others in this field is also described, and again the illustrations are excellent. Considering the amount of *exact* information that can nowadays be gleaned from an adequate X-ray examination, it is curious that radiological investigation in obstetrics is not used more frequently in this country.

All the Authors in the volume have appended extensive bibliographies to their sections, and there is a good general index.

The Editors are to be congratulated upon the excellent selection and quality of the illustrations, the general lay-out and readableness of the work, and, in short, the production of a British text-book that is certain to be highly esteemed both at home and abroad.

St. Thomas's Hospital Reports. Edited by Prov. O. L. V. S. de WESSELOW and C. MAX PAGE, assisted by N. R. BARRETT, J. ST. C. ELKINGTON, and A. J. WRIGLEY. Second Series, Vol. II. Royal 8vo. Pp. 271, illustrated. 1937. St. Thomas's Hospital. 10s. (7s. 6d. to subscribers).

THE present volume of *St. Thomas's Hospital Reports* contains articles relating to most branches of practice and scientific endeavour. Many are of no more than passing concern to surgical readers, but among the most important and interesting are some by non-surgical writers. Letheby Tidy contributes a remarkably complete and informative monograph on glandular fever, a disease rare enough to be unfamiliar, yet common enough to demand consideration in the differential diagnosis of conditions as far apart as mumps, diphtheria, septic lymphadenitis, Hodgkin's disease, and the exanthemata. In the first paper in the volume John Oliver discusses the scientific basis and practical value of the many serodiagnostic methods for the recognition of cancer in its pre-clinical stage, particularly the Bendien test and its modifications, making available for the surgeon information on many matters on which he is often consulted, or in any case feels that he should be able to form an intelligent opinion. Of interest to the gastric surgeon is a discussion on diet and the treatment of peptic ulcer by G. W. Hearn. He emphasizes the dangers of deficiency and alkalosis inherent in the standard methods of treatment by diet and drugs, and records his experience with the balanced high-caloric diet of Meulengracht; he is not convinced of its superiority as a routine treatment, but he has found that, in cases with severe hæmorrhage, the results have been superior to those obtained by the older methods.

Of the contributions by surgeons, the most striking in its wealth of detail and excellent figures is that by T. Pomfret Kilner on the technique of repair in cleft palate and hare lip. Whether such descriptions are wise is outside the purpose of this review. The post-graduate can more easily get detailed instruction how to undertake the most complicated operations that he is quite unfitted to perform, than he can get the practical experience that will make him safe in much simpler ones. Having studied this masterly paper, which is in fact a treatise on the operative surgery of this region, such a one might well be tempted to enter a field whose principles and performance can only be mastered by years of painstaking apprenticeship.

Mitchiner, Bailey, and Price compare the results of conservative and radical surgery in cancer of the breast, to the advantage of the former. They point out that, over a period of twenty years at St. Thomas's Hospital, the last ten of which are reviewed in their article, the late survivals were as numerous after a limited as after a radical operation, whereas the mortality, suffering, and deformity associated with the first are much less. These conclusions are so opposed to the experience of surgeons who can remember the dreadful days when limited surgery was the rule in breast cancer, and who still see the results of local operations done by correspondence-course surgeons, that they are unlikely, as the authors themselves suggest in the quotation from Isaiah that forms their text, to be accepted at once; but coming as they do from such an unimpeachable though iconoclastic source, they will receive the most serious consideration.

Gastric surgery is ably treated in three articles—on congenital pyloric stenosis, by Jewesbury and Max Page; on secondary gastrojejunal ulceration, by Romanis, and on perforating peptic ulcers, by Boggan. All three repay careful study, being examples of that most useful form of research, the analysis of results. One statement in the last—that "in spite of the unanimity of opinion amongst St. Thomas's surgeons, this is not shared by workers in other countries"—is delightfully reminiscent of the famous poster "Storm in the Channel. Continent Isolated."

Other articles of first-rate interest to the surgeon are those on the results of surgery in Graves' disease, by Goadby; on imperfect descent of the testes, by Mimpriss; on the results of operations for femoral hernia, by Fergusson; and on the control of infection in resection of the large bowel, by Nevin. Communications on subjects allied to surgery are those on the preparation of patients for operation, by Wilson; on the value of anæsthetic records, by Nosworthy; on the surgical treatment of ostosclerosis, by Howarth; and on the diagnosis and treatment of cancer of the lung, by Tchaperoff. This volume maintains the high standard of its predecessors, and is worth a place in any surgical library.

Innere Medizin in der Chirurgie. By Doz. Dr. med. habil. H. FRH. V. KRESS, Berlin, and Dr. med. W. KITTLER, Munich. Bd. 59 of *Neue deutsche Chirurgie*. $10\frac{1}{2} \times 7\frac{1}{8}$. Pp. 144 + viii. 1938. Stuttgart: Ferdinand Enke. RM. 10; bound, RM. 11.80.

THE need for closer co-operation between physicians and surgeons is felt as much in Germany as in this country. This book, which represents the collaboration of a physician and a surgeon, is a careful attempt to correlate the facts of medical practice with those of surgical. In the main it is an attempt to estimate the bearing of various abnormalities of the vital organs upon the expediency and outlook in any surgical operation. In the first place abnormalities of the circulation are considered and the various factors which go to make up cardiac insufficiency are evaluated. In the same way the bearing of diabetes, jaundice, and pulmonary disorders are considered. Its compact size and clear enunciation of principles will make it a most useful book of reference for the surgeon.

A Synopsis of the Diagnosis of the Acute Surgical Diseases of the Abdomen. By JOHN A. HARDY, B.Sc., M.D., F.A.C.S., El Paso, Texas. $8\frac{3}{4} \times 5\frac{1}{4}$ in. Pp. 345, with 92 illustrations. 1938. London: Henry Kimpton. 20s. net.

IN the preface it is made clear that this book is intended as a work of reference for those general practitioners in America who are called upon to do acute abdominal surgery.

It is pointed out that laboratory investigations play a very small part in the diagnosis of acute abdominal disease, and that one must rely upon clinical study and experience. To this end one is given an exhaustive description of the clinical picture of all the acute abdominal emergencies, and also of a considerable number of the subacute and chronic conditions with which they may be confounded. Many little-known signs and abstruse clinical methods are included.

The illustrations are good. They consist of diagrammatic drawings, which face the portion of the text that they illustrate. There is an admirable anatomical introduction.

The importance of a thorough general examination of the patient to exclude diseases of the chest and of the central nervous system is very properly emphasized.

The arrangement of the descriptive matter is a little unusual—the introductory chapter on general physical examination precedes that dealing with the history. "Physical Examination" precedes and is separated from "Signs and Symptoms" in each case. Inspection, palpation, percussion, and auscultation do not occur in their familiar order. We see no advantage in this unusual arrangement.

Aspiration of peritoneal fluid for pathological investigation is advocated in doubtful cases. It is pointed out that only a fraction of a drop is required. This procedure seems to present a number of practical difficulties, but is an interesting suggestion.

The volume contains a wealth of information and represents an exhaustive review of the subject. We can heartily recommend it to those for whom it is intended.

Hæmorrhoids. By MARION C. PRUITT, M.D., F.R.C.S. Ed., F.A.C.S., President, American Proctologic Society; Associate in Surgery, Emory University School of Medicine, Atlanta, Georgia; etc. 10 × 7½ in. Pp. 170, with 73 illustrations, including 7 in colour. 1938. London: Henry Kimpton. 18s. net.

THIS excellent monograph endeavours to assess the comparative values of the various methods advocated for the treatment of hæmorrhoids and their suitability to any given type of case—an objective in the attainment of which the author has exercised throughout a wise and judicious discrimination.

The anatomy of the anus, anal canal, and rectum is clearly and concisely described and excellently illustrated; indeed, the illustrations throughout the book constitute a most pleasing feature, and the seven coloured plates are of real artistic merit. For local infiltration anæsthesia during hæmorrhoidectomy the author finds a solution of 1–1000 nupercaine extremely satisfactory, and for topical application an ointment of the same drug in a strength of 1 per cent. For injection of the hæmorrhoids he uses a 12 per cent solution of carbolic acid in glycerin and water, of which 3 to 6 min. should be injected into the centre of each hæmorrhoidal mass: all the internal hæmorrhoids are injected at each treatment and the average interval between treatments is seven days. Recurrence of hæmorrhoids occurs in from 15 to 20 per cent within five years following injection treatment, and from 5 to 8 per cent following operation.

In the operative treatment Pruitt prefers excision and suture of each internal hæmorrhoid. The technique of this and of other recognized procedures is well described and illustrated. Errors in the text are very few, though in the description of *Fig.* 73 it is obvious that "inferior" hæmorrhoidal should read "superior".

Hernia: Anatomy, Etiology, Symptoms, Diagnosis, Differential Diagnosis, Prognosis, and the Operative and Injection Treatment. By LEIGH F. WATSON, M.D., Member of Attending Staff of California Lutheran Hospital and Methodist Hospital of Southern California, Los Angeles. Second edition. Large royal 8vo. Pp. 591, with 281 illustrations. 1938. London: Henry Kimpton. 31s. 6d. net.

THE first edition of this treatise was favourably reviewed in this JOURNAL in October, 1924. The main factor calling for a second edition is the increasing use in America of the modern injection treatment of certain types of reducible hernia—a method which the author's personal experience has convinced him is of value and of great economic importance in industry. Eight complete chapters are devoted to its general consideration and to its application to inguinal hernia, while reference thereto is also made under the headings of the other hernial types—in all about 100 pages of text. Much of the historical material of the first edition and the descriptions of many operations not now in general use have, of necessity, been omitted, the actual number of pages being some 70 less, although the illustrations number 50 more. Not only is the injection treatment very thoroughly described, but it is so excellently illustrated that any surgeon wishing to give it a trial will have no difficulty in understanding the technique, though it appears obvious that considerable practice is necessary to become expert. It would seem that this method has not received the attention it deserves in this country, whereas, on the contrary, in one of the American States (Minnesota) nearly 90 per cent of all industrial hernia claims that are approved by the Industrial Commission are offered the injection treatment. Moreover, there are many individuals with relaxed inguinal rings who, the author states, are almost certain to develop hernia at some time in their lives; "the injection treatment offers them a preventive means of cure and assures them against loss of their place in industry".

All the common and uncommon types of hernia are so completely and efficiently dealt with in this comprehensive treatise that it must long remain the standard work in this special branch of surgery.

The Foot. By NORMAN C. LAKE, M.D., M.S., D.Sc. (Lond.), F.R.C.S. (Eng.), Senior Surgeon and Lecturer on Surgery, Charing Cross Hospital; Surgeon, Bolingbroke Hospital; Director of Studies, London Foot Hospital; etc. Second edition. 8¾ × 5½ in. Pp. 366 + viii, with 113 illustrations. 1938. London: Baillière, Tindall & Cox. 12s. 6d. net.

IN his preface to the second edition the author states that it is not written for anyone in particular. It is certainly useful to the orthopædic surgeon, the student, the general practitioner, the chiropodist, and the masseur; but it would be more useful to the last named if the necessity for securing mobility and methods of re-educating position and use were described and emphasized. In fact, this defect might now be given as the chief one in an otherwise excellent production. Chapters have been added on Congenital Club-foot and on Foot Problems. The former is adequate without being of value to the specialist; the latter gives problems illustrative of the difficulties experienced in arriving at a true diagnosis—in this it is to be feared the author has been only partly successful. In the chapter on Examination of the Foot, there is an interesting account of attempts to estimate the distribution of pressure upon different parts of the sole on standing and walking, experiments which will ultimately lead to valuable knowledge. Among minor points that are open to criticism are that under Sprains of the Ankle attention has not been drawn to the effects of injuring the three different fasciculi of the external lateral ligament; and that under Flat-foot a very admirable account is vitiated by criticism of the present orthodox views of orthopædic surgeons, based upon the views of one particular surgeon who is known to be very unorthodox.

Diagnostik und therapeutische Indikationsstellung bei den chirurgischen Erkrankungen der Harnorgane. By Professor Dr. PETER JANSSEN, Düsseldorf. 9½ × 6¼ in. Pp. 316 + v. 1938. Berlin: Julius Springer. RM. 15; bound, RM. 16.80.

THE need for a simple exposition of the means of diagnosis and of the possibilities of treatment of the diseases of the urinary organs has become important as specialization in these subjects has advanced. The present work is a clearly written description of these diseases, together with matters of examination and diagnosis, which admirably fulfils this object. We should, however, have thought that the subject would have lent itself to illustration by line drawings, which would have made the book much more attractive and useful.

Die Röntgentiefentherapie. By Professor HANS HOLFELDER, Frankfurt A.M. Large royal 8vo. Pp. 341 + viii, with 164 illustrations and 1 coloured plate. 1938. Leipzig: George Thieme. RM. 27; bound, RM. 29.

THIS book is a publication of the annual lectures given in Frankfurt in May, 1937. It contains a special description of the methods of the Frankfurt school, and the progress of the radiotherapy institute. It is not meant to cover the whole ground of the subject, several common types of cancer receiving no mention.

The histology of tumours and of normal tissues at intervals after irradiation is discussed by Engiemann, and constitutes a very valuable part of the book.

The chapter on carcinoma of the stomach represents a definite advance in this most difficult branch of the subject. In cases formerly regarded as inoperable, the new procedure is surgical removal of the main growth as far as possible, followed by irradiation. This method does appear to hold out some hope, however slender—5.5 per cent of these inoperable cases are symptom-free at the end of three years, and 4 per cent at the end of five years.

The authors confirm the general conclusion that the combination of surgery and irradiation has also definitely improved their results in carcinoma of the breast. Dr. Weisswange, who has written this section, considers that post-operative irradiation should not be postponed for longer than four weeks after the operation; the optimum interval appears to be about fourteen days. This section of the book is particularly well illustrated; the diagrams showing the setting up of the patient and the distribution of the dosage in the tissues are instructive.

There is an interesting chapter on the effect on tumours of variations in the gradation of dosage. On these experiments are based the numerous tables showing the dosage used in the Frankfurt school. One wonders whether the rigid adherence to pre-arranged rhythm of dosage advocated here gives better results in all cases than a variation of the dosage according to the biological state of the tumour as observed during the course of treatment.

The book, as might be expected from the work of several authors, is somewhat uneven. The collaboration between the surgeon and the radiotherapist, the importance of which is

fully recognized in some sections, might have been stressed more in others. For example, in the section on pituitary tumours, the method of treatment and the results are stated, but no help is given to the radiotherapist as to when he should call in the aid of the cerebral surgeon. But, on the whole, the book is a stimulating account of recent work of one of the most important centres in Europe, and should be read by all who are interested in radiotherapy. Workers in this and other countries will look forward with eagerness to the volume which Professor Holfelder has promised to publish next year, dealing with the treatment of tumours arising in other situations.

The Treatment of Clinical and Laboratory Data. An Introduction to Statistical Ideas and Methods for Medical and Dental Workers. By DONALD MAINLAND, M.B., Ch.B., D.Sc. (Edin.), Professor of Anatomy, Dalhousie University, Halifax, Nova Scotia, Canada. Demy 8vo. Pp. 340 + xii, with 23 illustrations. 1938. Edinburgh: Oliver & Boyd Ltd. 15s. net.

"ANY one with even a rudimentary idea of the effects of chance will, if he reads medical literature, observe that in a great deal of it there is inadequate recognition and treatment of questions of chance, of the allowance to be made for small samples, and for natural variation from sample to sample of the same material, whether that material is composed of blood specimens, patients with paralysis, or animals subjected to drugs . . . The numerous books on the subject, including those on medical statistics, often give too much and too complicated detail for clinical workers, and commonly fail to show that statistical ideas are of any use to the practitioner."

The author has set himself to meet this deficiency, and the present volume is the outcome of his labours. He believes that "the method of exposition by working from examples to general principles and technique" is that which will appeal most readily to the average reader, and he follows this method throughout. As a consequence algebraical formulæ are reduced to a minimum.

The book is an admirable introduction to the methods of statistical reasoning. The main problems of statistics are successively presented, and at the end of each chapter a guide is given to further reading. The examples are chosen from subjects of ordinary clinical experience, and in the statistical discussion which follows, the reader need never lose sight of the problem with which he is familiar. Thus, for example, in the interpretation of significance by the x^2 method, the thoughts of the reader are firmly fixed on the occurrence of septicæmia after treatment of wounds by Lister's method, as compared with that after treatment by ordinary dressings. The interpretation of the results obtained gives the author scope for an admirable exposition of the meaning of *not significant* and the meaning of *significant*, and incidentally introduces the notion of association, and, though it is not explicitly stated, of fiduciary probability.

It is impossible in a short review to deal with all the points which seem to the reviewer to be especially attractive, but it should be emphasized that, as in the case of the above example, the interpretation of the meaning of the various numbers or values obtained by statistical methods, is discussed with a fullness and clearness which is rare in statistical literature. The non-mathematical reader, by the familiarity of the example chosen, should have no difficulty in understanding the argument, and even the expert will find modes of expression which may be useful to him. The book concludes with an admirable chapter on the preparation of data and of results for publication.

A Manual of Surgery for Nurses. By CHARLES WELLS, M.B., F.R.C.S., Hon. Surgeon with charge of Out-patients, Royal Southern Unit of the Royal Liverpool United Hospital; etc. Cr. 8vo. Pp. 409 + viii, with 159 illustrations. 1938. Edinburgh: E. & S. Livingstone. 10s. 6d. net, postage 6d.

THIS is a new book, which was probably written primarily for the nurses in Liverpool taught by the author, but owing to its clearness and freshness it will doubtless have a very much wider field. In a comparatively short compass the Author has succeeded in giving a wonderfully complete account of the main outline of surgery and operative technique required to be known by the nurses. Both the text and the illustrations are admirably clear, whilst the author has shown good judgment in his selection of subjects.

Surgical Nursing and After-treatment. By H. C. RUTHERFORD DARLING, M.D., M.S., F.R.C.S., Surgeon, Prince Henry Hospital, New South Wales Hospital (Langton Clinic), and New South Wales Masonic Hospital, Sydney. Sixth edition. $7\frac{1}{2} \times 5$ in. Pp. 714 + xii, with 206 illustrations. 1938. London: J. & A. Churchill Ltd. 9s. net.

IT is hardly necessary to say much about a book which has reached its sixth edition in eleven years. It is clearly the outcome of careful thought and experience of one who has not hesitated to seek the co-operation of his colleagues, both surgical and nursing. We think that the book would be improved by the omission of many of the illustrations of rather obsolete splints (e.g., *Figs.* 58, 67, 71) and by giving a more generous depiction of plaster-of-Paris methods.

Illustrations of Anatomy for Nurses. By E. B. JAMIESON, M.D., Senior Demonstrator and Lecturer, Anatomy Department, University of Edinburgh. $8 \times 6\frac{1}{4}$ in. Sixty-two plates, 59 of which are from the Author's *Illustrations of Regional Anatomy*. 1938. Edinburgh: E. & S. Livingstone. 7s. 6d. net, postage 6d.

THIS consists of 62 plates, most of them in colour, selected from the author's larger work on regional anatomy. As in the larger work, it is so bound that the plates can be detached separately. It will be a very welcome addition to a nurse's library.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

A Text-book of Surgical Pathology. By C. F. W. ILLINGWORTH, M.D., F.R.C.S. (Edin.), Lecturer in Clinical Surgery, University of Edinburgh; Senior Assistant Surgeon, Edinburgh Western Hospital; and B. M. DICK, M.B., F.R.C.S. (Edin.), Lecturer in Clinical Surgery, University of Edinburgh; Assistant Surgeon, Edinburgh Western Hospital. Third edition. 10 × 6¼ in. Pp. 727 + viii, with 299 illustrations. 1938. London; J. & A. Churchill Ltd. 36s. net.

Kollaterale Entzündungszustände (sog. akute Knochenatrophie und Dystrophie der Gliedmassen) in der Unfallheilkunde. By Dr. PAUL SUDECK. Heft 24 of *Monatsschrift für Unfallheilkunde und Versicherungsmedizin* (zur Verth). 9½ × 6¼ in. Pp. 68, with 44 illustrations. 1938. Berlin: F. C. W. Vogel. RM. 6.40.

On the Cause of Gastric Ulcers. An Experimental Study. By Dr. R. BRUMMELKAMP. In English. 9¾ × 6¼ in. Pp. 86, with 39 illustrations, most of which are plates. 1938. Amsterdam: N. V. Noord-Hollandsche Uitgeversmaatschappij. Fl. 3.50.

The Hypothalamus: Morphological, Functional, Clinical and Surgical Aspects. By W. E. LE GROS CLARK, D.Sc., F.R.C.S., F.R.S.; JOHN BEATTIE, M.D., D.Sc.; GEORGE RIDDOCH, M.D., F.R.C.P.; and NORMAN M. DOTT, F.R.C.S. Edin. 10 × 7½ in. Pp. 212 + xii, with over 100 illustrations. Published for The William Ramsay Henderson Trust. Edinburgh: Oliver & Boyd. 12s. 6d. net.

The Surgical Treatment of Hypertension. By GEORGE CRILE, edited by AMY ROWLAND. 9½ × 6 in. Pp. 239, with 52 illustrations. 1938. Philadelphia and London: W. B. Saunders Co. Ltd. 18s. net.

A Short Practice of Surgery. By HAMILTON BAILEY, F.R.C.S. (Eng.), Surgeon, Royal Northern Hospital, London; Surgeon and Urologist, Essex County Council; Surgeon, Italian Hospital., etc.; and R. J. MCNEILL LOVE, Surgeon, Royal Northern and Metropolitan Hospitals; etc. Fourth edition. 8½ × 5½ in. Pp. 996 + viii, with 818 illustrations, of which 109 are coloured. 1938. London: H. K. Lewis & Co. Ltd. 28s. net.

Clinical Roentgenology of the Digestive Tract. By MAURICE FELDMAN, M.D., Assistant Professor of Gastroenterology, University of Maryland, etc. 9 × 6 in. Pp. 1014 + xvi, with 358 illustrations. 1938. London: Baillère, Tindall & Cox. 45s. net.

Surgeon Extraordinary. The Life of J. B. Murphy. By LOYAL DAVIS, M.D., M.S., Ph.D., Professor of Surgery and Chairman of the Division of Surgery in North-Western University, Illinois. With a Foreword by A. J. Cronin. 8⅝ × 5⅝. Pp. 287. 1938. London: George G. Harrap & Co. Ltd. 8s. 6d. net.

Cancer, with special reference to Cancer of the Breast. By R. J. BEHAN, M.D., Dr. Med. (Berlin), F.A.C.S., Cofounder and formerly Director of the Cancer Department of the Pittsburg Skin and Cancer Foundation, Pittsburgh, Pa. 10½ × 6¾. Pp. 844 + xv, with 168 illustrations. 1938. London: Henry Kimpton. 42s. net.

Röntgendiagnostik der Knochen und Gelenkkrankheiten. By Professor Dr. ROBERT KIENBÖCK, Vienna. *Heft* 5, Rheumatoide Gelenkstuberkulose. 10½ × 7½ in. Pp. 451–1049, with 610 illustrations. 1938. Berlin and Vienna: Urban & Schwarzenberg. RM.60.